Evaluation of the Temporal Profile of Primary Headaches

Submitted in partial fulfillment of the requirements towards the conferment of

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of

THE TAMILNADU

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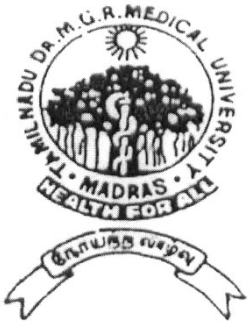
CHENNAI, TAMILNADU



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CERTIFICATE

This is to certify that this dissertation entitled **"**Evaluation of the Temporal Profile of Primary Headaches**"** submitted by **Dr. M. Radha** appearing for **D.M.,** Degree examination in **August 2009** is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I Dr. M.Radha do solemnly affirm that this dissertation titled "Evaluation of the Temporal Profile of Primary Headaches" is a done by me at Institute of Neurology, Madras Medical College & Govt. General Hospital,
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The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M., degree in Neurology.**

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Title of the work "Evaluation of the Temporal profile of primary headaches"

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting which is held on 31st March at 2 P.M in Government General Hospital, Deans, Chamber, Chennai-3.

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Contents

Introduction

Headache is the most common complaint of civilized human species world wide. It has the dubious distinction of being the earliest recognized symptom of a wide spectrum of diseases. Headache has been referred to in many ancient literatures. The earliest reference dates back to a *papyrus* (ancient medical text book) as old as 3500 BC in the tomb of *Thebes* that mentions the king in the tomb had sufferred all his life of a sickness of half head. A poem recovered from a Babylonian ruin written in Sumeria in 3000BC roughly translates as

The sick eyed says not…….
I am sick eyed…..
The sick headed not …….
I am sick headed…

Hippocrates (460-377BC) wrote a description of a disease of the hemicrania that relieved on vomiting. This work of dissertation has been done with an aim at documenting the different types of headache, their clinical presentations, diagnostic modalities and responses to prophylaxis, who presented to the Headache clinic of Institute of Neurology, Government General Hospital, Chennai during a one year period.

Aims and Objectives

This study is aimed to

1. To Classify and Study the incidence of various types of Primary Headache
2. Study the Clinical Profiles of each type of Primary Headache
3. Study the Neuro-physiological patterns in Migraine
4. Study the various patterns of responses to prophylaxis in Migraine.

Review of Literature

Headache or ***Cephalalgia*** is a symptom of a number of different conditions of the head and neck, caused by benign or at times a medical emergency that ranks among the most common pain complaints. One in three persons suffers a severe headache at some point in life and in US, headache causes on an average 112 million bed ridden days per year and morbidity

driven economic losses of $13 billion / year.

History

The earliest references date back to a papyrus (3500 BC) in the tomb of Thebes that mentions about the king in the tomb suffering of half head. Headache with neuralgia is referred to by Egyptians (1200 BC). Hippocrates (460-377BC) described the visual aura in migraine and its relief through vomiting[1]. **Aretaeus of Cappadocia** (2nd century AD) described a unilateral headache associated with vomiting, with headache-free intervals, and is credited as the "discoverer" of migraines.[2] **Galenus of Pergamon** used the term "hemicrania" (half-head) and thought there was a connection between the stomach and the brain, as nausea and vomiting often accompanied an attack.[2] In the Medieval Ages migraine was treated with hot irons, blood letting and even witchcraft. *Bibliotheca Anatomica, Medic, Chirurgica*, published in London (1712), describes five major types of headaches, including "Megrim", recognized later as classic migraine.[2]. Harold Wolffe (1950) developed the experimental approach to the study of headache and elaborated the vascular theory of migraine.[2]. Harold Wolffe (1950), enunciated a list of pain sensitive structures, both intracranial and extracranial namely: *Intracranial* - Circle of Willis & first few centimeters of its branches, meningeal (dural) arteries, large veins and dural venous sinuses and portions of dura near blood vessels; *Extracranial* – external carotid artery and its branches, scalp and neck muscles, skin and cutaneous nerves, cervical nerves and nerve roots, sinus, and teeth (via the V VII IX and X Cranial Nerves to CNS)[3][4]

The first recorded system that resembles the modern one was published by Thomas Willis, in *De Cephalagia* in 1672[4]. In 1787 Christian Baur classified headaches into idiopathic (primary headaches) and symptomatic (secondary headaches) and defined 84 categories. NIH developed a classification system in 1962 [5]. Presently headaches are classified by the International Headache Society's International Classification of Headache Disorders (ICHD), first edition published in 1988 and a second edition published in 2004 [6] and accepted by WHO.

The International Classification of Headache Disorders

The International Headache Society Classification with the diagnostic criteria for most headache disorders (ICHD-2) was published in 2004.[6] The classification uses numeric codes with one-digit diagnostic levels for 14 headache groups, the first four of which are labelled as primary headaches, while the group 5-12 are secondary headaches.[6]

I. Primary Headaches

1. Migraine
2. Tension-type headache (TTH)
3. Cluster headache and other trigeminal autonomic cephalalgias (TAC)
4. Other primary headaches

II. Secondary Headaches

5. Headache attributed to head and/or neck trauma - Head injury
6. Headache attributed to cranial or cervical vascular disorder including:

i. Intracerebral hemorrhage

ii. Subarachnoid hemorrhage

iii. Giant cell arteritis

7. Headache attributed to non-vascular intracranial disorder including:

   i. Idiopathic intracranial hypertension

   ii. Post dural puncture headaches

   iii. Ictal headache

8. Headache attributed to a substance or its withdrawal including:

   i. Medication overuse headaches

   ii. Medication or drug withdrawal headaches

   iii. Hang overs

9. Headache attributed to infection including: Meningitis

10. Headache attributed to disorder of homoeostasis

11. Headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures

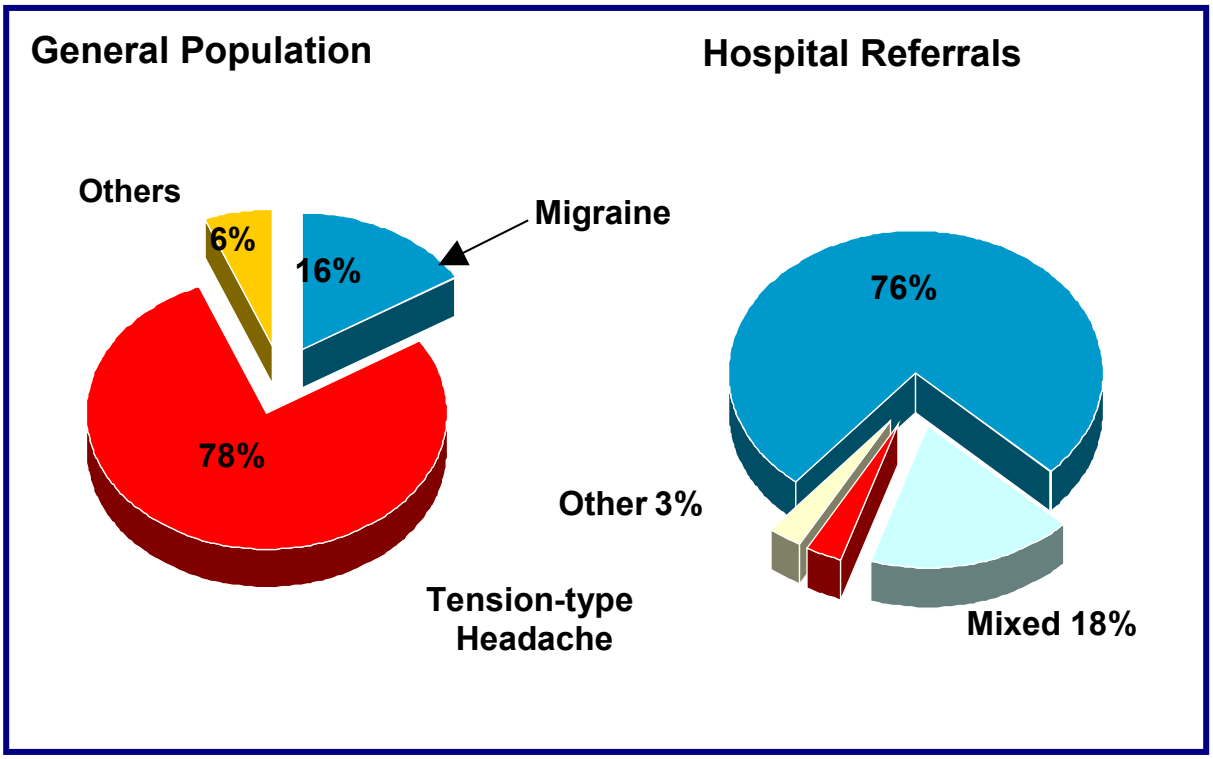
12. Headache attributed to psychiatric disorder

13. Cranial Neuralgias and Central Causes of facial pain

14. Others

Epidemiology of Headache Disorders

The Tension type headache is considered the most common form of headache in the general population with a prevalence of for nearly 80% while the prevalence of migraine is pegged at 16% in various international studies. In contrast, migraine is a more common form of headache reported in clinical practice. This variance is attributed to self treatment of tension type headaches by the general population.[7] A substantial group of patients, conform to the diagnostic criteria of more than one type of headache – these cases have been grouped under mixed headaches. The tension migraine or tension vascular headache is one common example.[7].



1. Migraine

Migraine is a common disabling primary headache disorder with a high prevalence, socio-economic and personal impacts, that is ranked by the World Health Organization as number 19 among all diseases world-wide causing disability.[8]

International Headache Society Classification of Migraine

1.1 Migraine without aura

1.2 Migraine with aura

1.2.1 Typical aura with migraine headache

1.2.2 Typical aura with non-migraine headache

1.2.3 Typical aura without headache

1.2.4 Familial hemiplegic migraine (FHM)

1.2.5 Sporadic hemiplegic migraine

1.2.6 Basilar-type migraine

1.3 Childhood periodic syndromes that are commonly precursors of migraine

1.3.1 Cyclical vomiting

1.3.2 Abdominal migraine

1.3.3 Benign paroxysmal vertigo of childhood

1.4 Retinal migraine

1.5 Complications of migraine

1.5.1 Chronic migraine

1.5.2 Status migrainosus

1.5.3 Persistent aura without infarction

1.5.4 Migrainous infarction

1.5.5 Migraine-triggered seizures

1.6 Probable migraine

1.6.1 Probable migraine without aura

1.6.2 Probable migraine with aura

1.6.5 Probable chronic migraine

International Headache Society Diagnosis Criteria for Migraine

1.1 Migraine without aura: This is a clinical syndrome characterised by headache with specific features and associated symptoms previously termed as common migraine and hemicrania simplex. This is characterised by recurrent headache disorder manifesting in attacks lasting 4-72 hours, with typical headache of unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia. The diagnostic criteria include: (A) At least 5 attacks fulfilling criteria B-D; (B) Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated); (C) Headache has at least two of the following characteristics: 1. unilateral location, 2. pulsating quality, 3. moderate or severe pain intensity, 4. aggravation by or causing avoidance of routine physical activity (*eg*, walking or climbing stairs); (D) During headache at least one of the following: 1. nausea and/or vomiting, 2. photophobia and phonophobia; and (E) Not attributed to another disorder

1.2 Migraine with aura: This entity previously termed classic or classical migraine, ophthalmic, hemiparaesthetic, hemiplegic or aphasic migraine, migraine accompagnée, complicated migraine etc, is a recurrent disorder of reversible focal neurological symptoms that develops gradually over 5-20 minutes and last for less than 60 minutes,. Headache with features of migraine without aura usually follows the aura symptoms, and less commonly, headache lacks migrainous features or is completely absent. The aura is a complex of neurological symptoms that occurs just before or at the onset of migraine headache. Most patients with migraine have exclusively attacks without aura. Many patients who have frequent attacks with aura may also have attacks without aura. Diagnostic criteria include [A] At least 2 attacks fulfilling criterion B [B] Migraine aura fulfilling criteria B and C for one of the subforms 1.2.1-1.2.6 & [C] Not attributed to another disorder. Premonitory symptoms occur hours to a day or two before a migraine attack (with or without aura), that include various combinations of fatigue, difficulty in concentrating, neck stiffness, sensitivity to light or sound, nausea, blurred vision, yawning and pallor. Auras with similar feature are also described in

other well-defined headache types like cluster headache. The relationship between aura and headache is not fully understood. Studies have demonstrated that patients with visual auras occasionally have symptoms in the extremities. Conversely patients with symptoms in the extremities virtually always also suffer visual aura symptoms. A distinction between migraine with visual aura and hemiparaesthetic migraine is hence not recognised in the present IHS classification [6].

1.2.1 Migraine with typical aura: Typical aura consists of visual and/or sensory and/or speech symptoms. Gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is associated with a headache fulfilling criteria for Migraine without aura. Diagnostic criteria include (A) At least 2 attacks fulfilling criteria B–D; (B) Aura consisting of at least one of the following, but no motor weakness: 1. fully reversible visual symptoms including positive features (eg, flickering lights, spots or lines) and/or negative features (ie, loss of vision), 2. fully reversible sensory symptoms including positive features (ie, pins and needles) and/or negative features (ie, numbness), 3. fully reversible dysphasic speech disturbance; (C) At least two of the following: 1. homonymous visual symptoms and/or unilateral sensory symptoms 2. at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes, 3. each symptom lasts ≥5 and ≤60 minutes; (D) Headache fulfilling criteria B-D for 1.1 Migraine without aura begins during the aura or follows aura within 60 minutes; (E) Not attributed to another disorder. This is the most common migraine syndrome associated with aura, the diagnosis of which is evident with careful history alone, but rarely carotid dissection, AV malformation and seizure may mimic this condition. Aura include visual aura, the most common type that often presents as a fortification spectrum or scotoma without positive phenomena; sensory aura, that presents as pins and needles moving slowly from the point of origin or numbness and speech disturbances like dysphasia. Symptoms usually follow one another in succession beginning with visual, then sensory symptoms and dysphasia.

1.2.2 : Typical aura with non-migraine headache: Typical aura consisting of visual and/or sensory and/or speech symptoms with gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is associated with a headache that does not fulfil criteria for 1.1 Migraine without aura. Diagnostic criteria includes all patterns as for Migraine with typical aura from A – C and a Headache that does not fulfil the criteria B-D set for 1.1 *Migraine without aura,* that begins during the aura or follows aura within 60 minutes. In the absence of headache fulfilling the criteria for 1.1 *Migraine without aura*, precise diagnosis of aura and its distinction from more serious diseases like transient ischemic attack becomes more important.

1.2.3: Typical aura without headache: Typical aura consisting of visual and/or sensory symptoms with or without speech symptoms. Gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is not associated with headache. A small number of patients have typical aura without headache are commonly patients with Typical aura with migraine who on becoming older have lost migraine characteristics, even though auras continue. Diagnostic criteria includes all patterns as for Migraine with typical aura from A – C without Headache during the aura or following aura within 60 minutes.

1.2.4: Familial hemiplegic migraine (FHM): Migraine with aura including motor weakness and at least one first- or second-degree relative has migraine aura including motor weakness. Specific genetic subtypes of FHM have been identified: (1) FHM1 - mutations in CACNA1A gene on chromosome 19 (2) FHM2 - mutations in the ATP1A2 gene on chromosome1, of these FHM1 very often has basilar-type symptoms in addition to the typical aura while headache is virtually always present. During FHM1 attacks, disturbances of consciousness (sometimes including coma), fever, CSF pleocytosis and confusion can occur. FHM1 attacks can be triggered by (mild) head trauma. In approximately 50% of FHM1 families, chronic progressive cerebellar ataxia occurs independently of the migraine attacks. FHM2 is very often mistaken for epilepsy, and (unsuccessfully) treated as such.

1.2.5 Sporadic hemiplegic migraine: Migraine with aura including motor weakness without first or second-degree relative having an aura including motor weakness. Epidemiological studies have shown that sporadic cases occur with approximately the same prevalence as familial cases. The attacks have the same clinical characteristics as those in FHM. Sporadic cases always require neuroimaging and other tests to rule out other cause. A

lumbar puncture is also necessary to rule out pseudomigraine with temporary neurological symptoms and lymphocytic pleocytosis. This condition is more prevalent in males and often associated with transient hemiparesis and aphasia.

1.2.6 Basilar-type migraine: Previously termed as Basilar artery migraine or basilar migraine, this entity is migraine with aura symptoms clearly originating from the brainstem and/or from both hemispheres simultaneously affected, but no motor weakness. Diagnostic criteria include: A. At least 2 attacks fulfilling criteria B-D; B. Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness: [1] dysarthria [2] vertigo [3] tinnitus [4] hypacusia [5] diplopia [6] visual symptoms simultaneously in both temporal and nasal fields of both eyes [7] ataxia [8] decreased level of consciousness [9] simultaneously bilateral paraesthesias; C. At least one of the following: [1] at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes [2] each aura symptom lasts ≥5 and ≤60 minutes; D.Headache fulfilling criteria B-D for 1.1 *Migraine without aura* begins during the aura or follows aura within 60 minutes; E. Not attributed to another disorder. Basilar-type attacks are mostly seen in young adults, many patients of whom report attacks with typical aura. In such case the code for both disorders need to be given. Patients with 1.2.4 FHM have basilar-type symptoms in 60% of cases. Therefore, 1.2.6 *Basilar-type migraine* should be diagnosed only when no motor weakness occurs. Many of the symptoms listed under criterion B are subject to misinterpretation as they may occur with anxiety and hyperventilation. Originally the terms *basilar artery migraine* or *basilar migraine* were used but, since involvement of the basilar artery territory is uncertain the term *basilar-type migraine* is preferred.

1.3 Childhood periodic syndromes that are commonly precursors of migraine.

1.3.1 Cyclical vomiting: Recurrent episodic attacks, usually stereotypical in the individual patient, of vomiting and intense nausea. Attacks are associated with pallor and lethargy. There is complete resolution of symptoms between attacks. Cyclical vomiting is a self-limiting episodic condition of childhood, with periods of complete normality between episodes. The clinical features of this syndrome resemble those found in association with migraine headaches, and multiple threads of research over the last years have suggested that cyclical vomiting is a condition related to migraine.

1.3.2 Abdominal migraine: An idiopathic recurrent disorder seen mainly in children and characterised by episodic midline abdominal pain manifesting in attacks lasting 1-72 hours with normality between episodes. The pain is of moderate to severe intensity and associated with vasomotor symptoms, nausea and vomiting. Pain is severe enough to interfere with normal daily activities. Children may find it difficult to distinguish anorexia from nausea. The pallor is often accompanied by dark shadows under the eyes. In a few patients flushing is the predominant vasomotor phenomenon. Most children with abdominal migraine will develop migraine headache later in life.

1.3.3 Benign paroxysmal vertigo of childhood: This probably heterogeneous disorder is characterised by recurrent brief episodic attacks of vertigo occurring without warning and resolving spontaneously in otherwise healthy children.

1.4 Retinal migraine: Repeated attacks of monocular visual disturbance, including scintillations, scotomata or blindness, associated with migraine headache.

1.5 Complications of migraine

1.5.1 Chronic migraine: Migraine headache occurring on 15 or more days per month for more than 3 months in the absence of medication overuse. Diagnostic criteria: [A] Headache fulfilling criteria C and D for 1.1 Migraine without aura on ≥15 days/month for >3 months. [B] Not attributed to another disorder. Most cases of chronic migraine start as 1.1 Migraine without aura and therefore, may be regarded as a complication of episodic migraine. As chronicity develops, headache tends to lose its attack-wise (episodic) presentation although it has not been clearly demonstrated that this is always so. Therefore, the default rule is to code such patients according to the antecedent migraine subtype plus probable chronic migraine plus probable medication-overuse headache.

1.5.2 Status migrainosus: A debilitating migraine attack lasting for more than 72 hours. Diagnostic criteria: [A] The present attack in a patient with 1.1 Migraine without aura is typical of previous attacks except for its duration [B] Headache has both of the following features: (1) unremitting for >72 hours (2) severe intensity [C] Not attributed to another disorder.

1.5.3 Persistent aura without infarction: Aura symptoms persist for more than 1 week without radiographic evidence of infarction. Diagnostic criteria: [A].The present attack in

a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for >1 week [B]. Not attributed to another disorder. Persisting aura symptoms are rare but well documented. They are often bilateral and may last for months or years. Reliably effective treatment is not known though acetazolamide and valproic acid have helped in a few cases. It is important to exclude posterior leukoencephalopathy by diffusion MRI and Migrainous infarction.

1.5.4 Migrainous infarction: One or more migrainous aura symptoms associated with an ischaemic brain lesion in appropriate territory demonstrated by neuroimaging. Diagnostic criteria: [A] The present attack in a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for >60 minutes [B] Neuroimaging demonstrates ischaemic infarction in a relevant area. [C] Not attributed to another disorder. Ischaemic stroke in a migraine sufferer may be categorised as cerebral infarction of other cause coexisting with migraine, cerebral infarction of other cause presenting with symptoms resembling migraine with aura, or cerebral infarction occurring during the course of a typical migraine with aura attack. Only the last fulfils criteria for 1.5.4 Migrainous infarction. Increased risk for stroke in migraine patients has been demonstrated in women under age 45 in several studies. Evidence for an association between migraine and stroke in older women and in men is inconsistent.

1.5.5 Migraine-triggered seizure: A seizure triggered by a migraine aura. Diagnostic criteria: [A] Migraine fulfilling criteria for 1.2 Migraine with aura [B] A seizure fulfilling diagnostic criteria for one type of epileptic attack occurs during or within 1 hour after a migraine aura. Migraine and epilepsy are highly comorbid conditions probably sharing the same pathophysiology, but the nature of their association is unclear. Migralepsy is the term used when a seizure occurs during or within 1 hour of a typical migraine aura attack. Reversible brain MRI abnormalities have been reported in a patient with migraine-triggered seizure, possibly due to supratentorial focal cerebral edema. Electroencephalogram (EEG) findings are usually normal interictal, although various abnormalities, mainly diffuse slowing, have been reported in migraineurs. [23]

Symptom	Migraine	Epilepsy
Duration of aura	15–60 min	Brief, often <1 min
Automatisms	Unusual	Frequent for complex

		partial seizures
Gastrointestinal aura	Abdominal pain (rare) Nausea (common)	‘‘Butterflies’’—rising epigastric sensation
Visual disturbances	Positive/negative	Complex visual phenomenon
Paresthesias	Common (5–60 min)	Common (seconds to minutes)
Altered consciousness	Usually responsive	Often unresponsive
Olfactory	Very uncommon	More common
Aphasia	Uncommon	Common
D_ej_a vu	Rare	Common

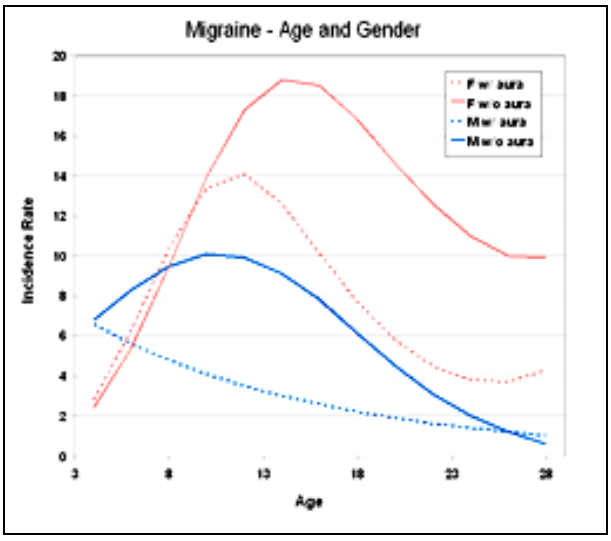
Migraine–epilepsy syndromes include (1) **Benign epilepsy of childhood with occipital paroxysms (BOEP)** - BOEP is a clinical syndrome characterized by visual symptoms followed by a partial seizure and postictal migraine. The EEG reveals occipital spikes. A rare syndrome of childhood (mean age of onset 7.5 years), it accounts for less than 5% of epilepsy in children.[24] BOEP has features common to both epilepsy and migraine. The seizures usually begin with visual symptoms, including amaurosis, elementary visual hallucinations (phosphenes) and complex visual hallucinations, or visual illusions, including micropsia, metamorphopsia, or palinopsia. The visual symptoms are often followed by hemiclonic complex partial seizures or generalized tonic–clonic seizures. Following the seizure, approximately 25 to 40% of the patients develop migraine-like headaches. The interictal EEG is characterized by normal background activity and distinct occipital discharges. The occipital spikes typically have a high voltage (200–300 *l*V), diphasic morphology, and a unilateral or bilateral occipital and posterotemporal distribution. The spikes disappear with eye opening and reappear 1 to 20 seconds after eye closure. Occipital spikes are not specific for BOEP. They have been reported in persons with migraine, and in children under 4, they may not be associated with epilepsy or any other defined disorder. Occipital spikes can also be seen in other disorders, including myoclonic, absence, and photosensitive epilepsies as well as in coeliac disease. (2) **Benign rolandic epilepsy:** Benign rolandic epilepsy is characterized by unilateral somatosensory or motor seizures and centrotemporal spikes. An association with migraine has been reported in some, but not all, studies.[25] Migraine prevalence in male controls (11.1%) was much higher than one would expect in boys between the ages of 6 and 15 years. Giroud and colleagues, in a controlled study, found that epilepsy with rolandic

paroxysms and migraine were associated. Migraine incidence was studied in four groups of patients: patients with centrotemporal epilepsy, patients with absence epilepsy, patients with partial epilepsy, and nonepileptic patients with a history of cranial trauma. Migraine was present in 62% of the patients with centrotemporal epilepsy, 34% of the patients with absence epilepsy, 8% of the patients with partial epilepsy, and 6% of the patients with cranial trauma.

1.6 Probable migraine: Previously termed as a migrainous disorder, attacks and/or headache missing one of the features needed to fulfil the criteria for the disorders coded above.

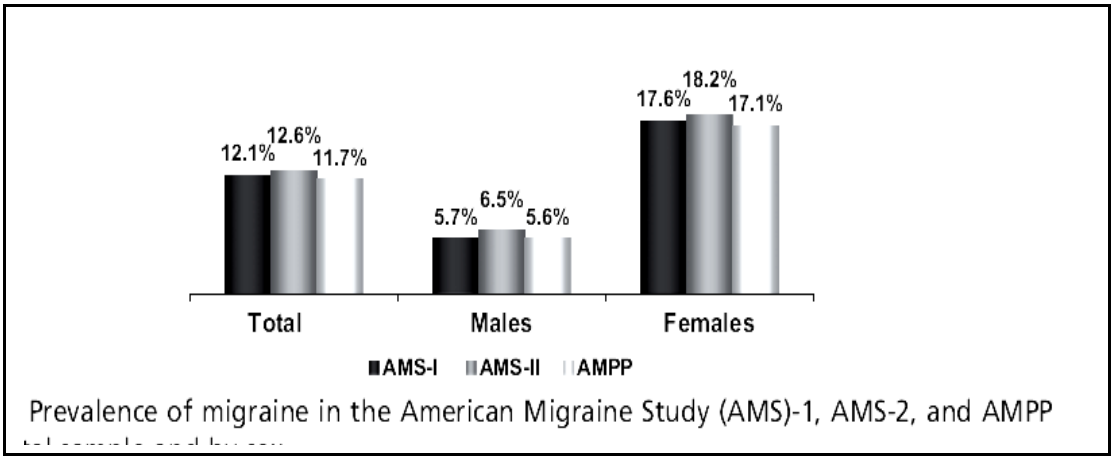
Demographics & Etiology

Epidemiology: According to IHS, migraine constitutes 16% of primary headaches. Migraine afflicts 10-20% of the general population. World - 15-20% of women and 10-15% of men suffer from migraine. In India, 15-20% of people suffer from migraine with an adult female: male ratio of 2:1.[8] [9] In childhood migraine, boys and girls are affected equally until puberty. In individuals older than 12 years, the prevalence increases in both males and females, and the incidence declines in individuals older than 40 years, except for women in perimenopause. The overall prevalence is higher in females than in males. The female-to-male ratio increases from 2.5:1 at puberty to 3.5:1 at age 40 years, after which it declines. The incidence of migraine with aura peaks in boys at around age 5 years and in girls at around age 12-13 years. The incidence of migraine without aura peaks in boys at age 10-11 years and in girls at age 14-17 years. The incidence of migraine in females of reproductive age has increased over the last 20 years, probably due to more awareness of the condition. In the United States, white women have the highest incidence of migraine, whereas Asian women have the lowest incidence. Moreover, low socioeconomic status is associated with migraine. Currently, 1 of 6 American women has migraine headaches.[10].



Age-Gender Incidence: Migraine is an extremely common condition which will affect 12–28% of people at some point in their lives. However this lifetime prevalence does not provide for how many patients there are with active migraine at any one time. The burden of migraine

in a population is assessed by looking at the one-year prevalence The third figure, which helps to clarify the picture, is the incidence that relates to the number of first attacks occurring at any given age and helps understanding of how the disease grows and shrinks over time. The one year prevalence of migraine ranges from 6–15% in adult men and from 14–35% in adult women.[11] These figures vary substantially with age: approximately 4–5% of children aged under 12 suffer from migraine, with little apparent difference between boys and girls. In the United States, three very large studies assessed the epidemiology of migraine in adults. The American Migraine Study-1 (AMS-1), collected information from 15,000 households representative of the US population in 1989.10 AMS-II, used virtually identical methodology 10 years later.11 Finally, the American Migraine Prevention and Prevalence study (AMPP) replicated, in its first research phase, the methods of AMS-I and AMS-II.[12] In these three very large studies, the prevalence of migraine was about 18% in women and 6% in men. There is then a rapid growth in incidence amongst girls occurring after puberty, [9][11] which continues throughout early adult life. By early middle age, around 25% of women experience a migraine at least once a year, compared with fewer than 10% of men.[1]After menopause, attacks in women tend to decline dramatically, so that in the over 70s there are approximately equal numbers of male and female sufferers, with prevalence returning to around 5%. At all ages, migraine without aura is more common than migraine with aura, with a ratio of between 1.5:1 and 2:1.[13] Incidence figures show that the excess of migraine seen in women of reproductive age is mainly due to migraine without aura.[13]



Prevalence of migraine in the American Migraine Study (AMS)-1, AMS-2, and AMPP

Thus in pre-pubertal and post-menopausal populations, migraine with aura is somewhat more common than amongst 15–50 year olds. There is a strong relationship between age, gender and type of migraine. The incidence of migraine is related to the incidence of epilepsy in

families, with migraine twice as prevalent in family members of epilepsy sufferers, and more common in epilepsy sufferers themselves[14].

Pathophysiology of Migraine

There are many schools of thought on the pathophysiology of migraine. The older vascular theory; is now considered secondary to brain dysfunction and has fallen out of favour and been replaced by newer theories.

Vascular theory: Migraines can begin when **blood vessels** in the brain contract and expand inappropriately. This may start in the occipital lobe, in the back of the brain, as arteries undergo spasm. The reduced flow of blood from the occipital lobe triggers the visual aura.[15],[16] When the constriction stops and the **blood vessels** dilate, the blood vessels become permeable and some fluid leaks out. This leakage is recognized by pain receptors in the **blood vessels** of surrounding tissue. In response, the body supplies the area with chemicals which cause inflammation. With each heart beat, blood passes through this sensitive area causing a throb of pain. Ray Wolffe et al (1940)[3] believed that intracranial vasoconstriction is responsible for the aura of migraine and that subsequent rebound vasodilatation and activation of perivascular nociceptive nerves resulted in headache, based on the observations that (1) extracranial vessels become distended and pulsatile during a migraine attack, (2) stimulation of intracranial vessels in an awake person induces headache, and (3) vasoconstrictors (eg, ergots) improve the headache, whereas vasodilators (eg, nitroglycerin) provoke an attack.[17]

Depolarization theory: A phenomenon known as **cortical spreading depression** can cause migraines.[18] In **cortical spreading depression**, **neurological activity** is depressed over an area of the **cortex** of the brain. This situation results in the release of **inflammatory** mediators leading to irritation of **cranial nerve** roots, most particularly the **trigeminal nerve**, which conveys the sensory information for the face and much of the head. This view is supported by **neuroimaging** techniques, which appear to show that migraine is primarily a disorder of the brain (neurological), not of the blood vessels (vascular). A spreading depolarization (electrical change) may begin 24 hours before the attack, with onset of the headache occurring around the time when the largest area of the brain is depolarized. A

French study in 2007, using the **PET** technique identified the **hypothalamus** as being critically involved in the early stages.[19]

Serotonin theory: **Serotonin**, a neurotransmitter, helps to control mood, pain sensation, sexual behaviour, sleep, as well as dilation and constriction of the blood vessels. Low **serotonin** levels in the brain may lead to a process of constriction and dilation of the blood vessels which trigger a migraine. **Triptans** activate serotonin receptors to stop a migraine attack.[18]

Neural theory: When an area in the brain stem become irritated, a migraine begins. In response to the irritation, the body releases chemicals which cause inflammation of the blood vessels. These chemicals cause further irritation of the nerves and blood vessels and results in pain. **Substance P** is one of the substances released with first irritation. Pain then increases because substance P aids in sending pain signals to the brain.[18]

Unifying theory: The theory encompasses both vascular and neural influences associated with stress that triggers changes in the brain, changes caused by serotonin release, blood vessels constriction, chemicals like substance P that irritate nerves and blood vessels causing pain.[18]

Cortical spreading depression: In 1944, Leao proposed this theory to explain the mechanism of migraine with aura. A migraine aura is due to well-defined wave of neuronal excitation in the cortical gray matter that spreads from its site of origin at the rate of 2-6 mm/min; followed by a wave of neuronal suppression in a similar fashion. The blood vessels in the area simultaneously dilate and constrict. Hence, migraine aura is considered a cortical event with definite and well-defined neuroelectrical basis. The neurochemical basis of the CSD is the release of potassium or the excitatory amino acid glutamate from neural tissue. This release depolarizes the adjacent tissue, which, in turn, releases more neurotransmitters, propagating the spreading depression. Positron emission tomography (PET) scanning demonstrates that blood flow is moderately reduced during a migrainous aura, but the spreading oligemia does not correspond to vascular territories. The oligemia itself is insufficient to impair function. The reason why these neurons are more excitable at a cellular level in certain patients is not entirely clear. Specific groups of patients with migraine have a

genetic defect leading to a lowered threshold for CSD, and this is called familial hemiplegic migraine (FHM). However, for the vast majority of patients, a clear metabolic or genetic defect that easily explains this neuronal excitability cannot be determined.[18],[19],[20]

Brainstem activation: PET scanning in patients having an acute migraine headache demonstrates activation of the contralateral pons, even after medications abort the pain. Weiler et al proposed that brainstem activation may be the initiating factor of migraine. Once the CSD occurs on the surface of the brain, H^+ and K^+ ions diffuse to the pia mater and activate C-fiber meningeal nociceptors, which releases proinflammatory neurochemicals (eg, calcitonin gene–related peptide) and plasma extravasation occurs. Therefore, a sterile, neurogenic inflammation of the trigeminovascular complex is present. Once the trigeminal system is activated, it stimulates the cranial vessels to dilate. The final common pathway to the throbbing headache is the dilatation of blood vessels.[19],[21][22]

Cutaneous allodynia: Burstein et al 2000 [23][24] described the phenomenon of cutaneous allodynia, in which secondary pain pathways of the trigeminothalamic system become sensitized during a migrainous episode. This observation further demonstrates that sensitization of central pathways in the brain mediates the pain of migraine, in addition to the previously described neurovascular events.

Dopamine Pathway: Some authors have proposed a dopaminergic basis for migraine. In 1977, Sicuteri postulated that a state of dopaminergic hypersensitivity is present in patients with migraine. Interest in this theory has recently been renewed [18]. A variety of prodromal symptoms (eg, yawning, irritability, nausea, vomiting) can be attributed to relative dopaminergic stimulation. Dopamine antagonists, such as prochlorperazine, completely relieve almost 75% of acute migraine attacks.

Magnesium Deficiency: Another theory proposes that deficiency of magnesium in the brain triggers a chain of events, starting with platelet aggregation and glutamate release and finally resulting in the release of 5-hydroxytryptamine, which is a vasoconstrictor.[18]

Clinical Features: Diagnosis, description, and triggers

The diagnosis of migraine is clinical in nature, based on criteria established by the IHS. The signs and symptoms of migraine vary among patients. Therefore, what a patient experiences before, during and after an attack cannot be defined exactly. The four phases of a migraine attack listed below are common but not necessarily experienced by all migraine sufferers. Additionally, the phases experienced and the symptoms experienced during them can vary from one migraine attack to another in the same migraineur:

1. The prodrome, which occurs hours or days before the headache.
2. The aura, which immediately precedes the headache.
3. The pain phase, also known as headache phase.
4. The postdrome.

Prodrome phase: Prodromal symptoms occur in 40–60% of migraineurs (migraine sufferers). This phase may consist of altered mood, irritability, **depression** or **euphoria**, **fatigue**, **yawning**, excessive sleepiness, craving for certain food (e.g. **chocolate**), stiff muscles (especially in the neck), constipation or diarrhea, increased urination, and other visceral symptoms[25][26] These symptoms usually precede the headache phase of the migraine attack by several hours or days, and experience teaches the patient or observant family how to detect that a migraine attack is near.

Aura phase: For the 20–30% of individuals who suffer migraine with aura, this aura comprises focal neurological phenomena that precede or accompany the attack. They appear gradually over 5 to 20 minutes and generally last fewer than 60 minutes. The headache phase of the migraine attack usually begins within 60 minutes of the end of the aura phase, but it is sometimes delayed up to several hours, and it can be missing entirely. Symptoms of migraine aura can be visual, sensory, or motor in nature.[27],[28]. Visual **aura** is the most common of the neurological events. There is a disturbance of vision consisting usually of unformed flashes of white and/or black or rarely of multicolored lights (**photopsia**) or formations of dazzling zigzag lines (**scintillating scotoma**; often arranged like the battlements of a castle, hence the alternative terms "fortification spectra" or "teichopsia". Some patients complain of blurred or shimmering or cloudy vision, as though they were looking through thick or **smoked glass**, or, in some cases, **tunnel vision** and **hemianopsia**. The somatosensory aura of

migraine consists of digitolingual or cheiro-oral **paresthesias**, a feeling of pins-and-needles experienced in the hand and arm as well as in the nose-mouth area on the same side. [29] [30] Paresthesia migrates up the arm and then extends to involve the face, lips and tongue. Other symptoms of the aura phase can include auditory or olfactory hallucinations, temporary **dysphasia**, **vertigo**, tingling or numbness of the face and extremities, and hypersensitivity to touch.[31][32]

Pain phase: The typical migraine headache is unilateral, throbbing, moderate to severe and can be aggravated by physical activity. Not all of these features are necessary. The pain may be bilateral at the onset or start on one side and become generalized, and usually alternates sides from one attack to the next. The onset is usually gradual. The pain peaks and then subsides, and usually lasts between 4 and 72 hours in adults and 1 and 48 hours in children. The frequency of attacks is extremely variable, from a few in a lifetime to several times a week, and the average migraineur experiences from one to three headaches a month. The head pain varies greatly in intensity.[27],[28] The pain of migraine is invariably accompanied by other features. **Nausea** occurs in almost 90 percent of patients, while vomiting occurs in about one third of patients. Many patients experience sensory hyperexcitability manifested by **photophobia**, **phonophobia** and seek a dark and quiet room. Blurred vision, nasal stuffiness, diarrhea, **polyuria**, **pallor** or sweating may be noted during the headache phase. There may be localized **edema** of the scalp or face, scalp tenderness, prominence of a vein or artery in the temple, or stiffness and tenderness of the neck. Impairment of concentration and mood are common. Lightheadedness, rather than true **vertigo** and a feeling of faintness may occur. The extremities tend to be cold and moist.[32]

Postdrome phase: The patient may feel tired, have head pain, cognitive difficulties, "hungover", gastrointestinal symptoms, mood changes and weakness.[33] Some people feel unusually refreshed or euphoric after an attack, whereas others note depression and **malaise**. Often, some of the minor headache phase symptoms may continue, such as loss of appetite, photophobia, and lightheadedness. For some patients, a 5 to 6 hour nap may reduce the pain, but slight headaches may still occur when standing or sitting quickly.

Defining Pain Severity: The IHS defines the intensity of pain with a verbal, four-point scale. [34]

Number	Name	Annotations
0	no pain	
1	mild pain	does not interfere with usual activities
2	moderate pain	inhibits, but does not wholly prevent usual activities
3	severe pain	prevents all activities

Triggers: A migraine trigger is any factor that, on exposure or withdrawal, leads to the development of an acute migraine headache. Triggers may be categorized as behavioral, environmental, infectious, dietary, chemical, or hormonal. Migraine attacks may be triggered by allergic reactions, bright lights, loud noises, and certain odours or perfumes, head trauma, physical or emotional stress, changes in sleep patterns, smoking or exposure to smoke, skipping meals, alcohol, menstrual cycle fluctuations, birth control pills, hormone fluctuations during the menopause transition, foods containing tyramine (red wine, aged cheese, smoked fish, chicken livers, figs, and some beans), monosodium glutamate (MSG) or nitrates (like bacon, hot dogs, and salami), other foods such as chocolate, nuts, peanut butter, avocado, banana, citrus, onions, dairy products, and fermented or pickled foods., medications (eg, nitroglycerin, histamine, reserpine, hydralazine, ranitidine, estrogen).[35] Sometimes the migraine occurs with no apparent "cause". The trigger theory supposes that exposure to various environmental factors precipitates, or triggers, individual migraine episodes. Migraine patients have long been advised to try to identify personal headache triggers by looking for associations between their headaches and various suspected trigger factors. Several studies have found some migraines are triggered by changes in weather. One study noted 62% of the subjects thought weather was a factor but only 51% were sensitive to weather changes.[35] Most likely to trigger a migraine were, temperature, high humidity, and changes in barometric pressure. One study found that for some migraineurs in India, washing hair in a bath was a migraine trigger. The triggering effect was probably due to delayed drying of the wet hair. [36]

Comorbidities of migraine: Migraine is associated with epilepsy (eg, benign rolandic epilepsy, benign childhood epilepsy), familial dyslipoproteinemias, hereditary hemorrhagic telangiectasia, Tourette syndrome, hereditary essential tremor, hereditary cerebral amyloid angiopathy, ischemic stroke (migraine with aura is a risk factor, with an odds ratio of 6), depression and anxiety, asthma, patent foramen ovale, and stroke. Epilepsy increases the relative risk of migraine by 2.4. The risk of posterior circulation strokes, especially cerebellar, is increased in migraineurs with aura. Female migraineurs, with or without aura, have an increased risk of deep white matter brain lesions. Several studies are currently investigating patent foramen ovale, which is seen in 18-21% of migraine patients[37]

Diagnostic Procedures

Neuroimaging is indicated for any of the following: [A] First or worst headache of the patient's life [B] Change in frequency, severity, or clinical features of the headache [C] Abnormal neurological examination [D] Progressive or new daily, persistent headache [E] Neurological symptoms that do not meet the criteria for migraine with typical aura or that themselves warrant investigation [E] Persistent neurologic deficit [F] Hemicrania that is always on the same side and associated with contralateral neurological symptoms [G] Inadequate response to routine therapy [H] Atypical clinical presentation. Neuroimaging studies that may be appropriate include CT scan and MRI. Other studies such as angiography, MRA, and MRV also may be indicated.[19] Agarwal et al [2008],[16] have described reversible MRI brain white matter abnormalities in a patient with migraine. Frishberg reviewed four CT scan studies, four MRI scan studies, and one combined MRI and CT scan study of 897 scans of patients who had migraine. These findings are combined with more recent reports of one CT scan study of 284 patients 36 and six studies of MRI scans of 444 patients for a total of 1625 scans of patients who had various types of migraine. Other than WMA, the studies showed no significant pathology except for four brain tumors (three of which were incidental findings) and one AVM (in a patient who had migraine and a seizure disorder). Sempere found a similarly low yield of 0.4%.

Electrophysiology: The electroencephalogram (EEG) was a standard test for evaluation of headaches in the pre-CT scan era. Gronseth and Greenberg [38] reviewed the literature from

1941 to 1994 on the usefulness of EEG in the evaluation of patients who had headache. Most of the articles had serious methodologic flaws. The only significant abnormality reported in studies with a relatively nonflawed design was prominent driving in response to photic stimulation (the H-response) in migraineurs who had a sensitivity ranging from 26%17 to 100%18 and a specificity from 80%19 to 91%.18 This finding, although interesting, is not necessary for the clinical diagnosis of migraine. Electrophysiological studies in migraine: a comprehensive review of their interest and limitations by A Ambrosini et al [2003] is indicated herein.[39]

Autonomic Function Tests: The rationale to study functions of the ANS in headache is primarily based on clinical observations. Changes of ANS functions are obvious in cluster headache with autonomic symptoms (so-called trigeminal autonomic cephalgias [TACs]) [40], [41],[42] and have been extensively investigated for these pains in the last 20 years. Changes of ANS function are suspected to be important in migraine but are less obvious in this headache. Kruszewski et al [2000][43] have extensively discussed the literature on headache with autonomic symptoms. In most studies small samples of patients have been investigated. Usually patients have been classified according to the diagnostic criteria of the International Headache Society and groups of patients were paralleled by healthy sex- and agematched control groups. Test–retest reliability has seldom been studied adequately and blinding has not been used. Most published studies focus on group differences in order to understand more about headache pathogenesis. The group differences have seldom been large, and it is generally not proven that ANS tests can help in diagnosing the headache disorder. Sensitivity and specificity is almost never discussed although some exceptions occur [44].

***Autonomic Function Tests in Migraine*:** Based on a literature review and an extensive investigation of patients with migraine, Thomsen, Olesen and coworkers conclude that 'Clear dysfunction of the sympathetic nervous system remains to be shown. Mild parasympathetic hypofunction with denervation supersensitivity may be present in migraine' [45],[46]. Results are variable, however. Boiardi et al. [44] reported for instance that the diastolic blood pressure response to sustained handgrip was impaired in 61% of migraine patients. In a recent study, no heart-rate variability differences between migraine patients and control subjects were found [47]. Micieli et al. [48] found increased basal pupillary diameter as well as increased light reflex contraction and dilatation velocities in migraineurs. Increased pupillary dilatation to phenylephrine eyedrops has also been found in

migraine[49],[50]. Recently it has been proposed that activation parasympathetic neurones in the pterygopalatine and otic ganglia leads to vasodilation of cranial blood vessels, release of inflammatory mediators and activation and/or sensitization of trigeminovascular afferents [51], [52]. Consistent with this idea appears to be that intranasal application of lidocaine may relieve migraine attacks[53],[54]. It is suggested that this treatment blocks impulse transmission in the pterygopalatine ganglion. However, this idea of involvement of the efferent parasympathetic neurones in (direct or indirect) activation or sensitization of trigeminovascular afferents needs better experimental verification. Furthermore, relief of migraine attacks by intranasal lidocaine requires a doubleblind, placebo-controlled approach. Finally, the latter results could probably also be explained by blockade of activity in trigeminovascular afferents passing through the pterygopalatine ganglion.

Procedures: Indications for LP include the following: [A] First or worst headache of a patient's life [B] Severe, rapid-onset, recurrent headache [C] Progressive headache [D] Atypical chronic intractable headache

Treatment

Conventional treatment focuses on three areas: trigger avoidance, abortive therapy, and prophylaxis. Drug therapy is considered *effective* to reduce the frequency or severity of migraine attacks by 50%.[55] Children and adolescents, are often first given drug treatment, but the value of diet modification should not be overlooked.

I. Abortive Therapy: The abortive treatment of migraine is divided into (1) Symptomatic treatment (2) Specific Anti-migraine drugs.

Non Specific Treatment of Migraine: There is a large group of disparate nonspecific acute medications for migraine, that include nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin (ASA), acetaminophen (APAP), analgesic combinations with or without caffeine, opioids, butalbital, isometheptene, also often in combinations, and medication classes including antihistamines, antinauseants, antiepilepsy drugs, and muscle relaxants. Aspirin-acetaminophen-caffeine (AAC) mixtures are Food and Drug Administration approved for migraine based on a randomized controlled trial (RCT) in which preselected patients who

were usually incapacitated (ie, required bed rest for their attacks) or who experienced vomiting 20% or more of the time were excluded. NSAIDs, such as naproxen, diclofenac and solubilized ibuprofen, also are superior to placebo in RCTs. Evidence for opioid use in acute migraine generally is negative. Jakubowski and colleagues found that even intermittent use of opioids interfered with successful therapeutic reversal of central sensitization and pain-free response in patients who had status migrainosus. Butalbital use had no specific evidence of efficacy and had an excessive risk for habituation and dependence. Prevention population-based study suggested that episodic use of butalbital was linked to transformation into chronic daily headache and medication overuse headache. It is wise to never prescribe butalbital for migraine. [56][58]

Specific Anti Migraine Treatment: (1) Triptans are serotonin -1B/D (5HT1B/D) agonists that work via the serotonin-1D receptors to inhibit CGRP and inflammatory peptide release in the meninges and prevent the pain signal from returning from the periphery to the trigeminal nucleus caudalis. They work via the 5HT1B receptor to constrict vessels dilated by CGRP. Because there are some 5HT1B receptors in coronary and other arteries, triptans are contraindicated in patients who have vascular disease. Triptans are classified into Group I and II. Group-I triptans are fast acting with response rates of two hours of medication eg- Sumatriptan, Zolmitriptan, Rizatriptan, Almotriptan and Eletriptan. While group II triptans have a minimally slower rate of activity at 4 hours eg: Naratriptan and Frovatriptan. Selecting the triptan necessitates determining how fast a migraine worsens. If onset is quick, a group I triptan is necessary. If emesis is common, a non oral formulation like sumatriptan SC or Zolimitriptan Nasal spray are preferred. Some evidence suggests a triptan-NSAID combination can reduce headache recurrence[57]. **(2) Ergots:** Until the introduction of sumatriptan in 1991, ergot derivatives were the primary oral drugs available to abort a migraine once it is established. Ergot drugs can be used either as a preventive or abortive therapy, though their relative expense and cumulative side effects suggest reserving them as an abortive rescue medicine. However, ergotamine tartrate tablets (usually with caffeine), though highly effective, and long lasting (unlike triptans), have fallen out of favour due to the problem of ergotism.[57] Both ergot and triptans have the same risk for vasoconstriction DHE is tolerated far better, is useful as an acute treatment of long migraines because of low recurrence, and can be used repetitively for status migrainosus and in detoxification from rebound. According to AAN guidelines, maximum dosing for DHE is 3 mg per day, up to 21 mg per week. Intranasal DHE dosing requires one spray (0.5 mg) into each nostril (without

sniffing) at the first sign of migraine, followed 15 minutes later by an additional spray into each[58] **Other agents: Steroids:** Based on a recent meta analysis a single dose of IV dexamethasone, when added to standard treatment, is associated with a 26% decrease in headache recurrence.[59] Amidrine, Duadrin, and Midrin is a combination of acetaminophen, dichloralphenazone, and isometheptene often prescribed for migraine headaches. Some studies have recently shown that these drugs may work better than sumatriptan for treating migraines.[9][57] Recently it has been found that calcitonin gene related peptides (CGRPs) play a role in the pathogenesis of the pain associated with migraine as triptans also decrease its release and action. CGRP receptor antagonists such as olcegepant and telcagepant are being investigated both in vitro and in clinical studies for the treatment of migraine.

II. Prophylactic Therapy

US evidence-based guidelines for preventive treatment of migraine include the following:

1. Recurring migraine that significantly interferes with the patient's daily routine despite acute treatment (eg, two or more attacks a month that produce disability that lasts 3 or more days, headache attacks that are infrequent but produce profound disability
2. Failure of, contraindication to, or troublesome side effects from acute medications
3. Overuse of acute medications
4. Special circumstances, such as hemiplegic migraine or attacks with a risk for permanent neurologic injury.
5. Frequent headaches (more than two a week) or a pattern of increasing attacks over time, with the risk for developing medication overuse headache.
6. Patient preference, that is, the desire to have as few acute attacks as possible

Only 13% of all migraineurs currently use preventive therapy to control their attacks. The principles of preventive prophylaxis are

1. Start the chosen drug at a low dose, and increase it slowly until therapeutic effects develop, the ceiling dose for the chosen drug is reached, or adverse events (AEs) become intolerable. Give each treatment an adequate trial. The full benefit of the drug may not be realized until 6 months have elapsed.
2. Set realistic goals. Success is defined as a 50% reduction in attack frequency, a significant decrease in attack duration, or an improved response to acute medication.
3. Set realistic expectations regarding AEs. The risk and extent of AEs vary greatly from patient to patient, and we presently have no way of predicting the presence or severity of AEs for an individual patient. Most AEs are self-limited and dose dependent, and

patients should be encouraged to tolerate the early AEs that may develop when a new medication is started.

4. Avoid acute headache medication overuse and drugs that are contraindicated because of coexistent or comorbid illnesses.
5. Re-evaluate therapy, and, if possible, taper or discontinue the drug after a sustained period of remission (6–9 months).
6. Be sure that a woman of childbearing potential is aware of any potential risks, and choose the medication that has the least potential for AE on a fetus.
7. To maximize compliance, involve patients in their own care. Take patient preferences into account when deciding between drugs of relatively equivalent efficacy and tolerability.
8. Consider comorbidity, which is the presence of two or more disorders whose association is more likely than chance.

The major medication groups for preventive migraine treatment include

1. Anticonvulsants
2. Antidepressants
3. Beta-adrenergic blockers
4. Calcium channel antagonists
5. Serotonin antagonists
6. Botulinum neurotoxins
7. Nonsteroidal anti-inflammatory drugs
8. Others - Riboflavin, Magnesium.

If preventive medication is indicated, the agent should be chosen from one of the first-line categories based on the drug's relative efficacy in double-blind placebo-controlled trials, its side effect profile, and the patient's preference, in addition to coexistent and comorbid conditions. Preventive treatment is often recommended for only 6 to 9 months; however, to date, no randomized placebo-controlled trials have been performed to investigate migraine frequency after the preventive treatment has been discontinued. Diener and colleagues12 assessed 818 patients who had migraine and were treated with topiramate for 6 months to see the effects of topiramate discontinuation. Although the number of migraine days did increase. These findings suggest that patients should be treated for 6 months, with the option to continue to 12 months.

Beta-Adrenergic Blockers: Beta—blockers, the most widely used class of drugs in

prophylactic migraine treatment, are approximately 50% effective in producing a greater than 50% reduction in attack frequency. Evidence has consistently demonstrated that non selective beta-blocker propranolol is significantly effective, selective b1-blockers metoprolol, atenolol, bisoprolol, nadolol, and timolol are also effective, while beta-blockers with intrinsic sympathomimetic activity (eg, acebutolol, alprenolol, oxprenolol, pindolol) are not effective for migraine prevention. Propranolol is effective for migraine prevention at a daily dose of 120 to 240 mg, but no correlation has been found between its dose and its clinical efficacy. [58] The action of beta—blockers is probably central and could be mediated by (1) inhibiting central b-receptors that interfere with the vigilance-enhancing adrenergic pathways, (2) interaction with 5—HT receptors (but not all effective beta—blockers bind to the 5—HT receptors), and (3) cross-modulation of the serotonin system. Propranolol inhibits nitric oxide (NO) production by blocking inducible NO synthase. Propranolol also inhibits kainate-induced currents and is synergistic with N-methyl- D-aspartate blockers, which reduce neuronal activity and have membrane-stabilizing properties. Contraindications to the use of beta-blockers include asthma and chronic obstructive lung disease, congestive heart failure, atrioventricular conduction defects, Raynaud's disease, peripheral vascular disease, and brittle diabetes.[59]

Antidepressants: Antidepressants consist of several different drug classes with different mechanisms of action. Only one member of the class of tricyclic anti-depressants (TCAs), (amitriptyline) has proved efficacy in migraine. Although the mechanism by which antidepressants work to prevent migraine headache is uncertain, it does not result from treating masked depression. Antidepressants are useful in treating many chronic pain states, including headache, independent of the presence of depression, and the response occurs sooner than the expected antidepressant effect. The antidepressants that are clinically effective in headache prevention inhibit noradrenaline and 5—HT reuptake or are antagonists at the 5—HT2 receptors. The TCA dose range is wide and must be individualized. Most TCAs are sedating. Start with a low dose of the chosen TCA at bedtime, except when using protriptyline, which should be administered in the morning. If the TCA is too sedating, switch from a tertiary TCA (eg, amitriptyline, doxepin) to a secondary TCA (eg, nortriptyline, protriptyline). [60] AEs are common with TCA use. Antimuscarinic AEs include dry mouth, a metallic taste, epigastric distress, constipation, dizziness, mental confusion, tachycardia, palpitations, blurred vision, and urinary retention, conduction abnormalities, especially in the elderly, and these patients should be carefully monitored or other agents should be considered. Start at a dose of 10 to 25 mg at bedtime. The usual effective dose for migraine

ranges from 25 to 200 mg. Nortriptyline, a major metabolite of amitriptyline, is a secondary amine that is less sedating than amitriptyline. Start at a dose of 10 to 25 mg at bedtime. The dosage ranges from 10 to 150 mg/d. Protriptyline is a secondary amine that is similar to nortriptyline. Start at a dose of 5 mg in the morning. The dosage ranges from 5 to 60 mg/d as a single or split dose. [61]

Calcium Channel Antagonists: Two types of calcium channels exist: calcium entry channels, which allow extracellular calcium to enter the cell, and calcium release channels, which allow intracellular calcium (in storage sites in organelles) to enter the cytoplasm. Calcium entry channel subtypes include voltage-gated, opened by depolarization; ligand gated, opened by chemical messengers, such as glutamate; and capacitative, activated by depletion of intracellular calcium stores. The mechanism of action of the calcium channel antagonists in migraine prevention is uncertain, but possibilities include inhibition of 5—HT release, neurovascular inflammation, or the initiation and propagation of cortical spreading depression. *Flunarizine*, a nonselective calcium channel antagonist with antidopaminergic properties, was superior to placebo in six of seven randomized clinical trials.[61] The dose is 5 to 10 mg given at night, women seem to need lower doses than men. The most prominent AEs include weight gain, somnolence, dry mouth, dizziness, hypotension, occasional extrapyramidal reactions, and exacerbation of depression. Because of its side effects, flunarizine should be considered as a second-line drug for migraine prevention, after beta-blockers. *Verapamil* was more effective than placebo in two of three trials, but both positive trials were small and dropout rates were high, rendering the findings uncertain. Rigorous randomized controlled trial evidence does not exist to support the use of verapamil for migraine. Nimodipine, nicardipine, diltiazem, and cyclandelate, other nonselective calcium channel antagonists, cannot be recommended for migraine prophylaxis.[61]

Anticonvulsants: Anticonvulsants are increasingly recommended for migraine prevention because of well-conducted placebo-controlled trials. With the exception of valproic acid, topiramate, and zonisamide, anticonvulsants may substantially interfere with the efficacy of oral contraceptives. ***Carbamazepine*** 600 to 1200 mg/d, may be effective in preventive migraine treatment but it is rarely used in clinical practice for this purpose.[61] ***Gabapentin*** (1800–2400 mg) showed efficacy in a placebo-controlled double-blind trial, the attack frequency was reduced by 50% in approximately one third of patients. The most common AEs were dizziness or giddiness and drowsiness. [61]***Valproic Acid*** is a simple 8-carbon, 2-chain fatty acid. Divalproex sodium is a combination of valproic acid and sodium valproate. Both are

effective as is an extended-release form of divalproex sodium. In 1992, Hering and Kuritzky evaluated the efficacy of sodium valproate in migraine treatment in a double-blind, randomized, crossover study. Sodium valproate was effective in preventing migraine or reducing the frequency, severity, and duration of attacks in 86.2% of 29 patients, whose attacks were reduced from 15.6 to 8.8 a month. Nausea, vomiting, and gastrointestinal distress are the AEs that occur most commonly; their incidence decreases, however, particularly after 6 months. Later,
tremor and alopecia can occur. Valproate has little effect on cognitive functions and rarely causes sedation. Valproate is teratogenic.[61] ***Topiramate****:* Two large, pivotal, multicenter, randomized, double-blind, placebo-controlled clinical trials assessed the efficacy and safety of topiramate (50, 100, and 200 mg/d) in migraine prevention. In the first trial, the responder rate (patients withR50%reduction in monthly migraine frequency) was 52% with topiramate, 200 mg/d (P<.001); 54% with topiramate, 100 mg/d (P<.001); and 36% with topiramate, 50 mg/d (P 5 .039), compared with 23% with placebo.The 200-mg dose was not significantly more effective than the 100-mg dose. The second pivotal trial54 had significantly more patients who exhibited at least a 50% reduction in mean monthly migraines in the groups treated with topiramate at a dosage of 50 mg/d (39%; P 5 .009), 100 mg/d (49%; P 5 .001), and 200 mg/d (47%; P 5 .001). The most common AE of topiramate is paresthesia; other common AEs are fatigue, decreased appetite, nausea, diarrhea, weight decrease, taste perversion, hypoesthesia, and abdominal pain. In the migraine trials, body weight was reduced an average of 2.3% in the 50-mg group, 3.2% in the 100-mg group, and 3.8% in the 200-mg group. The most common central nervous system AEs were somnolence, insomnia, mood problems, anxiety, difficulty with memory, language problems, and difficulty with concentration. Renal calculi can occur with topiramate use.[62] ***Lamotrigine:*** blocks voltage-sensitive sodium channels, leading to inhibition of neuronal glutamate release of glutamate. Chen and colleagues reported on two patients who had migraine with persistent aura-like visual phenomena for months to years. After 2 weeks of lamotrigine treatment, both had resolution of the visual symptoms. Although open-label studies have suggested that lamotrigine may have a select role in the treatment of patients with frequent or prolonged aura, results from a placebo-controlled study in migraine were negative. Steiner and colleagues59 compared the safety and efficacy of lamotrigine (200 mg/d) and placebo in migraine prophylaxis in a double-blind, randomized, parallel-group trial. Improvements were greater on placebo, and these changes, which were not statistically significant, indicate that

lamotrigine was ineffective for migraine prophylaxis. [62]***Other Drugs*** include AngiotensinS-Converting Enzyme Inhibitors and Angiotensin II Receptor Antagonists, Botulinum Toxin Type A, Medicinal Herbs and Vitamins Feverfew (Tanacetum parthenium) is a medicinal herb whose effectiveness has not been totally established.[62]

2. Tension-type headache (TTH)

Tension headaches, renamed **tension-type headaches** by the International Headache Society in 1988, are the most common type of primary headaches. Tension-type headaches account for nearly 90% of all headaches. Approximately 3% of the population suffers from chronic-tension type headache.[2] The International Headache Society (IHS) classification system of 1988, included tension-type headache as a category, further defined as either episodic or chronic. Whilst this type of headache was previously considered to be primarily psychogenic, a number of studies have appeared after the first edition of the ICHS that strongly suggest a neurobiological basis, at least for the more severe subtypes of tension-type headache.[6] Tension headaches were previously termed as - muscle contraction headache, psychomyogenic headache, stress headache, ordinary headache, essential headache, idiopathic headache and psychogenic headache. The division into *episodic* and *chronic* subtypes that was introduced in the first edition of the classification has proved extremely useful. The chronic subtype is a serious disease causing greatly decreased quality of life and high disability. The exact mechanisms of tension-type headache are not known.

I HS CLASSIFICATION OF TENSION-TYPE HEADACHES –ICHD -2

2.1 Infrequent episodic tension-type headache

- 2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness
- 2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness

2.2 Frequent episodic tension-type headache

- 2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness
- 2.2.2 Frequent episodic tension-type headache not associated with pericranial

tenderness

2.3 Chronic tension-type headache

2.3.1 Chronic tension-type headache associated with pericranial tenderness

2.3.2 Chronic tension-type headache not associated with pericranial tenderness

2.4 Probable tension-type headache

2.4.1 Probable infrequent episodic tension-type headache

2.4.2 Probable frequent episodic tension-type headache

2.4.3 Probable chronic tension-type headache

IHS DIAGNOSTIC CRITERIA FOR TENSION TYPE HEADACHES

2.1 Infrequent episodic tension-type headache:

Infrequent episodes of headache lasting minutes to days, the pain being typically bilateral, pressing or tightening in quality and of mild to moderate intensity, which does not worsen with routine physical activity, without nausea but photophobia or phonophobia may be present. Diagnostic criteria include (A) At least 10 episodes occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B-D (B) Headache lasting from 30 minutes to 7 days (C) Headache has at least two of the following characteristics: (1) bilateral location (2) pressing/tightening (non-pulsating) quality (3) mild or moderate intensity (4) not aggravated by routine physical activity such as walking or climbing stairs (D) Both of the following: (1) no nausea or vomiting (anorexia may occur) (2) no more than one of photophobia or phonophobia (E) Not attributed to another disorder. [6]

2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness: Diagnostic criteria includes (A) Episodes fulfilling criteria A-E for 2.1 Infrequent episodic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness: Diagnostic criteria includes: (A) Episodes fulfilling criteria A-E for 2.1 Infrequent episodic tension-type headache (B) No increased pericranial tenderness. Increased pericranial tenderness recorded by manual palpation is the most significant abnormal finding in patients with tension-type headache. The tenderness increases with the intensity and frequency of headache and is further increased during actual headache. The diagnostic value of EMG and pressure algometry is limited Palpation is a useful guide for the treatment

strategy. It also adds value and credibility to the explanations given to the patient.

2.2 Frequent episodic tension-type headache:

Frequent episodes of headache lasting minutes to days. The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, and it does not worsen with routine physical activity. There is no nausea but photophobia or phonophobia may be present. Diagnostic criteria include (A) At least 10 episodes occurring on ≥1 but <15 days per month for at least 3 months (≥12 and <180 days per year) and fulfilling criteria B-D (B) Headache lasting from 30 minutes to 7 days, (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity, 4. not aggravated by routine physical activity such as walking or climbing stairs, (D) Both of the following: 1. no nausea or vomiting (anorexia may occur), 2. no more than one of photophobia or phonophobia, (E) Not attributed to another disorder. Frequent tension-type headache often coexists with migraine without aura. Coexisting tension-type headache in migraineurs should preferably be identified by a diagnostic headache diary. The treatment of migraine differs considerably from that of tension-type headache and it is important to educate patients to differentiate between these types of headaches in order to select the right treatment and to prevent medication-overuse headache.

2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness:

Diagnostic criteria include: (A) Episodes fulfilling criteria A-E for 2.2 Frequent episodic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.2.2 Frequent episodic tension-type headache not associated with pericranial tenderness:

Diagnostic criteria include: (A) Episodes fulfilling criteria A-E for 2.2 Frequent episodic tension-type headache (B) No increased pericranial tenderness

2.3 Chronic tension-type headache

This is a disorder evolving from episodic tension-type headache, with daily or very frequent episodes of headache lasting minutes to days. The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, and it does not worsen with routine physical activity. There may be mild nausea, photophobia or phonophobia. Diagnostic criteria: (A) Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year)1 and fulfilling criteria B-D (B) Headache lasts hours or may be continuous (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity 4. not aggravated by

routine physical activity such as walking or climbing stairs (D) Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea, 2. neither moderate or severe nausea nor vomiting, (E) Not attributed to another disorder

When it is uncertain how many attacks fulfil one or other set of criteria it is strongly recommended to use a diagnostic headache diary prospectively. In many uncertain cases there is overuse of medication. When this fulfils criterion B for any of the subforms of 8.2 Medication-overuse headache, the default rule is to code for 2.4.3 Probable chronic tension-type headache plus 8.2.8 Probable medication-overuse headache. When these criteria are still fulfilled 2 months after medication overuse has ceased, 2.3 Chronic tension-type headache should be diagnosed and 8.2.8 Probable medication-overuse headache discarded. It should be remembered that some patients with chronic tension-type headache develop migraine-like features if they have severe pain and , conversely, some migraine patients develop increasingly frequent tension-type-like interval headaches, the nature of which remains unclear.

2.3.1 Chronic tension-type headache associated with pericranial tenderness: Diagnostic criteria: (A) Headache fulfilling criteria A-E for 2.3 Chronic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.3.2 Chronic tension-type headache not associated with pericranial tenderness: Diagnostic criteria: (A) Headache fulfilling criteria A-E for 2.3 Chronic tension-type headache (B) No increased pericranial tenderness

2.4 Probable tension-type headache

Patients meeting one of these sets of criteria may also meet the criteria for one of the subforms of 1.6 Probable migraine.

2.4.1 Probable infrequent episodic tension-type headache: Diagnostic criteria: (A) Episodes fulfilling all but one of criteria A-D for 2.1 Infrequent episodic tension-type headache (B) Episodes do not fulfil criteria for 1.1 Migraine without aura (C) Not attributed to another disorder.

2.4.2 Probable frequent episodic tension-type headache: Diagnostic criteria: (A) Episodes fulfilling all but one of criteria A-D for 2.2 Frequent episodic tension-type headache (B) Episodes do not fulfil criteria for 1.1 Migraine without aura (C) Not attributed to another disorder.

2.4.3 Probable chronic tension-type headache: Diagnostic criteria: (A) Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year) and fulfilling

criteria B-D, (B) Headache lasts hours or may be continuous (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity, 4.not aggravated by routine physical activity such as walking or climbing stairs. (D) Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea
2. neither moderate or severe nausea nor vomiting (E) Not attributed to another disorder but there is, or has been within the last 2 months, medication overuse fulfilling criterion B for any of the subforms of 8.2 Medication-overuse headache

Pathophysiology

Various precipitating factors may cause TTH in susceptible individuals [2]. One half of patients with TTH identify stress or hunger as a precipitating factor that include - Stress - Usually occurs in the afternoon after long stressful work hours, Sleep deprivation & Eyestrain, Uncomfortable stressful position and/or bad posture, Irregular meal time (hunger), and Caffeine withdrawal. Until recently it was believed that tension headaches were caused by muscle tension around the head and neck. Recent research has shown that tension headache patients do not have increased muscle tension.[63] Another theory is that the pain may be caused by a malfunctioning pain filter which is located in the brain stem. The view is that the brain misinterprets information, for example from the temporal muscle or other muscles, and interprets this signal as pain. One of the main neurotransmitters which is probably involved is serotonin.[64]. Recent studies of nitric oxide (NO) mechanisms suggest that NO may play a key role in the pathophysiology of CTTH.[64] The sensitization of pain pathways may be caused by or associated with activation of nitric oxide synthase (NOS) and the generation of NO. Patients with chronic tension-type headache have increased muscle and skin pain sensitivity, demonstrated by low mechanical, thermal and electrical pain thresholds. Hyperexcitability of centralnociceptive neurons (in trigeminal spinal nucleus, thalamus, and cerebral cortex) is believed to be involved in the pathophysiology of chronic tension-type headache.[64] Recent evidence for generalized increased pain sensitivity or hyperalgesia in CTTH strongly suggests that pain processing in the central nervous system is abnormal in this primary headache disorder. Moreover, a dysfunction in pain inhibitory systems may also play a role in the pathophysiology of chronic tension-type headache.[65]

Frequency: Tension-type headaches can be **episodic** or **chronic**[6] Episodic tension-type headaches are defined as tension-type headaches occurring fewer than 15 days a month, whereas chronic tension headaches occur 15 days or more a month for at least 6 months. Tension-type headaches can last from minutes to days, months or even years, though a typical tension headache lasts 4–6 hours

Epidemiology

Headaches account for 1-4% of all emergency department (ED) visits and are the ninth most common reason for a patient to consult a physician. Physicians classify 90% of headaches reported to them as muscle contraction or migraine headaches. No literature suggests that headache frequency is different in other regions of the world. A female preponderance exists, 14-17%, compared with 5-6% in males. All ages are susceptible, but most patients are young adults.[65]

- Approximately 60% of headache onset occurs in those older than 20 years.
- Headache onset is unusual in those older than 50 years.

Clinical Features

Pain onset in tension-type headache can have a throbbing quality and is usually more gradual than onset in migraines. Compared with migraines, tension-type headaches are more variable in duration, more constant in quality, and less severe.[6]

- IHS diagnostic criteria for tension-type headaches states that 2 of the following characteristics must be present:
    - Pressing or tightening (nonpulsatile quality)
    - Frontal-occipital location
    - Bilateral - Mild/moderate intensity
    - Not aggravated by physical activity
- Tension-type headache history is as follows:
    - Duration of 30 minutes to 7 days

- No nausea or vomiting (anorexia may occur)
- Photophobia and/or phonophobia
- Minimum of 10 previous headache episodes; fewer than 180 days per year with headache to be considered "infrequent"
- Bilateral and occipitonuchal or bifrontal pain - Pain described as "fullness, tightness/squeezing, pressure," or "bandlike/viselike"
- May occur acutely under emotional distress or intense worry & Insomnia, often present upon rising or shortly thereafter
- Muscular tightness or stiffness in neck, occipital, and frontal regions
- Duration of more than 5 years in 75% of patients with chronic headaches
- Difficulty concentrating
- No prodrome

Physical examination serves mainly to exclude the possibility of other headache causes.

- Vital signs should be normal.
- Normal neurologic examination
- Tenderness may be elicited in the scalp or neck, but no other positive physical exam findings should be noted.
- Pain should not be elicited over temporal arteries or positive trigger zones.
- Some patients with occipital tension headaches may be very tender when upper cervical muscles are palpated.
- Pain associated with neck flexion and stretching of paracervical muscles must be distinguished from nuchal rigidity associated with meningeal irritation.[66][67][68]

Laboratory Studies

Laboratory work should be unremarkable in cases of tension-type headache. Specific tests should be obtained if the history or physical examination suggests another diagnostic possibility. Head CT scan or MRI is necessary only when the headache pattern has changed recently, the headache cannot be clearly defined by the clinician as a common primary

headache disorder (that is not a cluster, migraine, or tension-type of headache), or neurologic examination reveals abnormal findings.[67]

Treatment

Nonsteroidal anti-inflammatory drugs (NSAIDs): These agents may alleviate headache pain by inhibiting prostaglandin synthesis, reducing serotonin release, and blocking platelet aggregation. Although the effects of NSAIDs in the treatment of headache pain tend to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include naproxen, ketoprofen, and ketorolac.[66]

Follow-Up

Physical therapy for patients with headache includes warm and cold packs, ultrasound, and electrical stimulation. Regular exercise, stretching, balanced meals, and adequate sleep is part of a headache prevention program. Trigger point injections, occipital nerve blocks, or changes that improve posture may be used.

Deterrence/Prevention: Deterrence and prevention of headache may include the following: Physical therapy, Biofeedback and relaxation therapy, Cervical traction and Injection of trigger points.

Complications: Complications of headache may include the following: Undue reliance on nonprescription caffeine-containing analgesics, Dependence on/addiction to narcotic analgesics, GI bleed from use of NSAIDs, Risk of epilepsy 4 times greater than that of the general population

Prognosis: Headache may become chronic if life stressors are not changed. Most cases are intermittent and do not interfere with work or normal life span.

3. Trigeminal autonomic cephalalgias

Cluster headache, nicknamed "**suicide headache**", is a neurological disease that involves, as its most prominent feature, an immense degree of pain. "Cluster" refers to the tendency of these headaches to occur periodically, with active periods interrupted by spontaneous remissions.[6]. The first complete description of cluster headache was given by the London neurologist Wilfred Harris in 1926. He named the disease Migrainous neuralgia [72]. Cluster headaches have been called by several other names in the past including Erythroprosopalgia of Bing, Ciliary neuralgia, Erythromelagia of the head, Horton's headache (named afterBayard T. Horton, an American neurologist), Histaminic cephalalgia, Petrosal neuralgia, sphenopalatine neuralgia, Vidian neuralgia, Sluder's neuralgia, and Hemicrania angioparalyticia.[73]. It affects approximately 0.1% of the population, and men are more commonly affected than women. The trigeminal autonomic cephalalgias share the clinical features of headache and prominent cranial parasympathetic autonomic features. Experimental and human functional imaging suggests that these syndromes activate a normal human trigeminal-parasympathetic reflex with clinical signs of cranial sympathetic dysfunction being secondary.

IHS CLASSIFICATION –ICHD -2

3.1 Cluster headache

3.1.1 Episodic cluster headache

3.1.2 Chronic cluster headache

3.2 Paroxysmal hemicrania

3.2.1 Episodic paroxysmal hemicrania

3.2.2 Chronic paroxysmal hemicrania (CPH)

3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)

3.4 Probable trigeminal autonomic cephalalgia

3.4.1 Probable cluster headache

3.4.2 Probable paroxysmal hemicrania

3.4.3 Probable SUNCT

IHS DIAGNOSTIC CRITERIA

3.1 Cluster headache

This entity presents with attacks of severe, strictly unilateral pain which is orbital, supraorbital, temporal or in any combination, lasting 15-180 minutes, occurring from once every other day to 8 times a day. The attacks are associated with one or more of the following, all of which are ipsilateral: conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis, eyelid oedema. Most patients are restless or agitated during an attack. Diagnostic criteria:

A. At least 5 attacks fulfilling criteria B-D

B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15-180 minutes if untreated

C. Headache is accompanied by at least one of the following:
   1) ipsilateral conjunctival injection and/or lacrimation, 2) ipsilateral nasal congestion and/or rhinorrhoea, 3) ipsilateral eyelid oedema, 4) ipsilateral forehead and facial sweating, 5) ipsilateral miosis and/or ptosis, 6) a sense of restlessness or agitation

D. Attacks have a frequency from one every other day to 8 per day

E. Not attributed to another disorder

Acute attacks involve activation of the posterior hypothalamic grey matter. Cluster headache may be inherited (autosomal dominant) in about 5% of cases. Attacks usually occur in series (cluster periods) lasting for weeks or months separated by remission periods usually lasting months or years. However, about 10-15% of patients have chronic symptoms without remissions. In a large series with good follow-up, 27% of patients had only a single cluster period. These should be coded as 3.1Cluster headache. During a cluster period, and in the chronic subtype, attacks occur regularly and may be provoked by alcohol, histamine or nitroglycerine. Pain is maximal orbital, supraorbital, temporal or in any combination of these sites, but may spread to other regions of the head. Pain almost invariably recurs on the same side during an individual cluster period. During the worst attacks, the intensity of pain is excruciating. Patients are usually unable to lie down and characteristically pace the floor. Age at onset is usually 20-40 years. For unknown reasons prevalence is 3-4 times higher in men than in women. Cluster headache with coexistent trigeminal neuralgia (cluster-tic syndrome): Some patients have been described who have both 3.1Cluster headache and 13.1Trigeminal neuralgia. They should receive both diagnoses. The importance of this observation is that

both conditions must be treated for the patient to be headache free.

3.1.1 Episodic cluster headache: Cluster headache attacks occurring in periods lasting 7 days to 1 year separated by pain-free periods lasting 1 month or longer. Diagnostic criteria: (A) Attacks fulfilling criteria A-E for 3.1Cluster headache. (B) At least two cluster periods lasting 7-365 days1 and separated by pain-free remission periods of ≥1 month.

3.1.2 Chronic cluster headache: Cluster headache attacks occurring for more than 1 year without remission or with remissions lasting less than 1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-E for 3.1 Cluster headache, (B) Attacks recur over >1 year without remission periods or with remission periods lasting <1 month. Chronic cluster headache may arise de novo (previously referred to as primary chronic cluster headache) or evolve from the episodic subtype (previously referred to as secondary chronic cluster headache). Some patients may switch from chronic to episodic cluster headache.

3.2 Paroxysmal hemicrania: Attacks with similar characteristics of pain and associated symptoms and signs to those of cluster headache, but they are shorter-lasting, more frequent, occur more commonly in females and respond absolutely to indomethacin. Diagnostic criteria: (A) At least 20 attacks fulfilling criteria B-D, (B) Attacks of severe unilateral orbital, supraorbital or temporal pain lasting 2-30 minutes, (C) Headache is accompanied by at least one of the following: 1. ipsilateral conjunctival injection and/or lacrimation, 2. ipsilateral nasal congestion and/or rhinorrhoea, 3. ipsilateral eyelid oedema, 4. ipsilateral forehead and facial sweating, 5. ipsilateral miosis and/or ptosis; (D) Attacks have a frequency above 5 per day for more than half of the time, although periods with lower frequency may occur; (E) Attacks are prevented completely by therapeutic doses of indomethacin (F) Not attributed to another disorder. There is no male predominance. Onset is usually in adulthood, although childhood cases are reported. *Paroxysmal hemicrania with coexistent trigeminal neuralgia (CPH-tic syndrome):* Patients who fulfil criteria for both 3.2 Paroxysmal hemicrania and 13.1 Trigeminal neuralgia should receive both diagnoses. The importance of this observation is that both conditions require treatment. The pathophysiological significance of the association is not yet clear.

3.2.1 Episodic paroxysmal hemicrania: Attacks of paroxysmal hemicrania occurring in periods lasting 7 days to 1 year separated by pain-free periods lasting ≥1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-F for 3.2 Paroxysmal hemicrania (B) At least two attack periods lasting 7-365 days and separated by pain-free remission periods of ≥1 month

3.2.2 Chronic paroxysmal hemicrania (CPH): Attacks of paroxysmal hemicrania occurring for >1 year without remission or with remissions lasting <1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-F for 3.2 Paroxysmal hemicrania Attacks recur over >1 year without remission periods or with remission periods lasting <1 month

3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT): This syndrome is characterised by short-lasting attacks of unilateral pain that are much briefer than those seen in any other TAC and very often accompanied by prominent lacrimation and redness of the ipsilateral eye. Diagnostic criteria: (A) At least 20 attacks fulfilling criteria B-D; (B) Attacks of unilateral orbital, supraorbital or temporal stabbing or pulsating pain lasting 5-240 seconds; (C) Pain is accompanied by ipsilateral conjunctival injection and lacrimation; (D) Attacks occur with a frequency from 3 to 200 per day; (E) Not attributed to another disorder. This syndrome was described after the publication of the first edition of The International Classification of Headache Disorders and has become well recognised in the last decade. Patients may be seen with only one of conjunctival injection or tearing, or other cranial autonomic symptoms such as nasal congestion, rhinorrhoea or eyelid oedema may be seen. 3.3 SUNCT may be a subform of A3.3 Short-lasting Unilateral Neuralgiform headache attacks with cranial Autonomic symptoms (SUNA), described in the appendix. The literature suggests that the most common mimics of 3.3 SUNCT are lesions in the posterior fossa or involving the pituitary gland.
SUNCT with coexistent trigeminal neuralgia: Patients have been described in whom there is an overlap between 3.3 SUNCT and 13.1 Trigeminal neuralgia. Such patients should receive both diagnoses. This differentiation is clinically difficult.

3.4 Probable trigeminal autonomic cephalalgia

Headache attacks that are believed to be a subtype of trigeminal autonomic cephalalgia but which do not quite meet the diagnostic criteria for any of the subtypes described above. Diagnostic criteria: Attacks fulfilling all but one of the specific criteria for one of the subtypes of trigeminal autonomic cephalalgia. Not attributed to another disorder. Patients coded as 3.4 Probable trigeminal autonomic cephalalgia or one of its subforms either have had an insufficient number of typical attacks or fail to fulfil one of the other criteria.

3.4.1 Probable cluster headache: Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-D for 3.1 Cluster headache (B) Not attributed to another disorder

3.4.2 Probable paroxysmal hemicrania: Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-E for 3.2 Paroxysmal hemicrania (B) Not attributed to another disorder

3.4.3 Probable short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing

Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-D for 3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) (B) Not attributed to another disorder

Epidemiology

Cluster headaches are diagnosed more often in men. The male-to-female ratio in cluster headache ranges from 4:1 to 7:1. It primarily occurs between the ages of 20 to 50 years.[74] This gap between the sexes has narrowed over the past few decades. Limited epidemiological studies have suggested prevalence rates of between 56 and 326 people per 100,000. Custer headaches, are more common away from the equator towards the poles.[75]

Pathophysiology

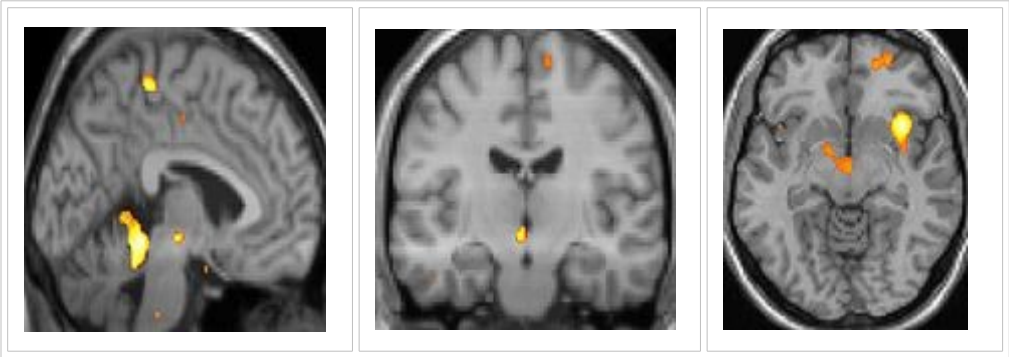
Cluster headaches classified as vascular headaches cause intense pain - caused by the dilation of blood vessels which creates pressure on the trigeminal nerve. While this process is the immediate cause of the pain, the etiology (underlying cause or causes) is not fully understood.[42]

Hypothalamic Abnormality: Among the most widely accepted theories is that cluster headaches are due to an abnormality in the **hypothalamus**; Dr Goadsbys' theory explains why cluster headaches frequently strike around the same time each day, and during a particular season, since one of the functions the hypothalamus performs is regulation of the **biological clock**.[74] **Metabolic** abnormalities have also been reported in patients. The hypothalamus is responsive to light / **day length and photoperiod**; **olfactory** stimuli, including **phero-mones** ; **steroids** including **sex steroids** and **corticosteroids**; neurally transmitted information arising in particular from the **heart**, **stomach**, **reproductive system**; **blood** borne stimuli including **leptin** **ghrelin**, **angiotensin**, **insulin**, **pituitary** **hormones**, **cytokines**, **blood plasma** concentrations of **glucose** and **osmolarity**, etc and **stress**.[76]

Genetics: There is a **genetic** component to cluster headaches, although no single gene has been identified as the cause. First-degree relatives of sufferers are more likely to have the condition than the population at large.[77] However, genetics appears to play a much smaller role in cluster headache than in some other types of headaches.

Triggers: **Nitroglycerin** (glyceryl trinitrate) can sometimes induce cluster headaches in sufferers in a manner similar to spontaneous attacks. Ingestion of **alcohol**, chocolate, exposure to **hydrocarbons** (petroleum solvents, perfume), heat, napping, nicotine may trigger cluster headaches.[78]

Neuroimaging



Functional MRI shows brain areas being activated during pain

The above fMRI pictures indicate the brain areas which are activated during pain only, compared to the pain free periods. These pictures show brain areas which are always active during pain in yellow/orange colour (called "pain matrix"). The area in the centre (in all three views) is specifically activated during cluster headache only. The bottom row Voxel-based morphometry (VBM) pictures show structural brain differences between cluster headache patients and people without headaches; only a portion of the hypothalamus is different. [79], [80]

Clinical Features

Cluster headaches are excruciating unilateral headaches,of extreme intensity. The duration of the common attack ranges from as short as 15 minutes to three hours or more. The onset of an attack is rapid, and most often without the preliminary signs that are characteristic of a migraine. However, some sufferers report preliminary sensations of pain in the general area of attack, often referred to as "shadows", that may warn them an attack is imminent.[74] Though the headaches are almost exclusively unilateral, there are many documented as cases of "side-shifting" between cluster periods, or, even rarer, simultaneously (within the same cluster period) bilateral headache. They are often initially mistaken for brain tumors

and multiple sclerosis until patients are treated with corticosteroids and then imaged. Trigeminal neuralgia can also bring on headaches with similar qualities. However, with Trigeminal neuralgia the pain is mostly located around the "cheek" area and is described as being more lance-like in quality. [74]

Pain: The degree of pain involved in cluster headaches is markedly greater than in other headache conditions, including severe migraines, and experts believe that it may be the most severe pain known to medical science. It has been described by female patients as being more severe than childbirth.[74] The McGill Pain Index can be used to rate levels of pain. The pain is lancinating or boring in quality, and is located behind the eye (periorbital) or in the temple, sometimes radiating to the neck or shoulder. Analogies frequently used to describe the pain are a red-hot poker inserted into the eye, or a spike penetrating from the top of the head, behind one eye, radiating down to the neck, or sometimes having a leg amputated without any anaesthetic.

Cyclical recurrence and regular timing: Cluster headaches are occasionally referred to as "alarm clock headaches", because of its ability to wake a person from sleep, and because of the regularity of its timing in that both the individual attacks and the clusters themselves can have a metronomic regularity; attacks striking at a precise time of day each morning or night is typical, even precisely at the same time a week later. This has prompted researchers to speculate an involvement of the brain's "biological clock" or circadian rhythm. [6][74]

Other symptoms include ptosis, conjunctival injection, lacrimation, rhinorrhea, facial blushing, swelling and sweating. These features are known autonomic symptoms. The attack is also associated with restlessness, aversion to bright lights and loud noise during the attack, nausea is comparatively rare. The neck is often stiff or tender with jaw or tooth pain[6][74].

Episodic or Chronic Cluster Headache: occur once or more daily, often at the same times each day, for a period of several weeks, followed by a headache-free period lasting weeks, months, or years. Cluster headaches occurring in two or more cluster periods lasting from 7 to 365 days with a pain-free remission of one month or longer between the clusters are considered episodic. If the attacks occur for more than a year without a pain-free remission of at least one month, the condition is considered chronic. Chronic clusters run continuously without any "remission" periods between cycles. The condition may change from chronic to

episodic and from episodic to chronic. Remission periods lasting for decades before the resumption of clusters are known to occur. Cluster headaches often go undiagnosed for many years, being confused with migraine or other causes of headache.[6][74]

Treatment

Medications to treat cluster headaches are classified as either abortives or prophylactics (preventatives). In addition, short-term transitional medications (such as steroids) may be used while prophylactic treatment is instituted and adjusted. With abortive treatments often only decreasing the duration of the headache and preventing it from reaching its peak rather than eliminating it entirely, preventive treatment is always indicated for cluster headaches, to be started at the first sign of a new cluster cycle.

Abortive treatment: During the onset of a cluster headache, some patients respond to rapid inhalation of pure oxygen (12-15 litres per minute in a non-rebreathing mask).[81][82] When used at the onset this can abort the attack in as little as 1 minute or as long as 10 minutes. Once an attack is at its peak, oxygen therapy appears to have little effect so most people have an oxygen provider close. Alternative first-line treatment is subcutaneous administration of triptan drugs, like sumatriptan and zolmitriptan. The injectable form of sumatriptan has been shown to abort a cluster headache within fifteen minutes in 96% of cases. Because of the rapid onset of an attack, the triptan drugs are usually taken by subcutaneous injection rather than by mouth. Beta blockers as a treatment has been tried. Recently, researchers have linked low testosterone as a possible cause of cluster headaches, which can be especially troublesome since the most effective pain medications for clusters, like morphine, reduce testosterone levels. Lidocaine and other topical anesthetics sprayed into the nasal cavity may relieve or stop the pain, Vaso-constrictors such as ergot compounds were also used. Other abortive remedies that work include ice, hot showers, cool or lukewarm water sprayed on the face around the sinus, temple, and ear areas, breathing cold air, caffeine, and drinking large amounts of water in the early stages of an attack. Hyperbaric oxygen therapy has been used successfully in treating cluster headaches though it was not shown to be more successful than surface oxygen[82].

Prophylactic Treatment: A wide variety of **prophylactic** medicines are in use, and patient response to these is highly variable. Current European guidelines suggest the use of

the **calcium channel blocker** **verapamil**ate a dose of at least 240 mg daily. **Steroids**, such as **prednisolone**, are also effective, with a high dose given for the first five days or longer before tapering down. **Methysergide**, **lithium** and **anticonvulsant** **topiramate** are recommended as alternative treatments. **Muscle relaxants** and atypical **anti-psychotics** have also been used. **Magnesium** supplements have been shown to be of some benefit in about 40% of patients. **Melatonin** has also been demonstrated to bring significant improvement in approximately half of episodic patients. **Other** neuropathic pain alleviating agents such as the **Tricyclic antidepressants** including **Amitriptyline** and **Nortriptyline** can also be used [82].

Materials and Methods

The patients registered at the **Headache Clinic**, **Institute of Neurology, Madras Medical College. Chennai 600003** during a one year period between the March 2007 and February 2008 were taken for this study. The clinical material was collected from the records of the headache Clinic - OPD case sheets and patient interviews with a detailed pre-prepared proforma [Appendix-1].

Inclusion Criteria

1. Patients registered at the Headache Clinic, Institute of Neurology, Madras Medical College. Chennai 600003 between 1st March 2007 and 28th February 2008
2. Patients in all age groups in both sexes, of any racial and socioeconomic denomination and profession were included
3. Patients were followed up for one year from the date of their registration and those who completed one year of follow up were included. All headaches were classified according to the International Headache Society Criteria 2004.

Exclusion Criteria

1. Patients with systemic, metabolic, traumatic disorders and or radiological findings that were documented to be directly or indirectly related to the causation of headache were excluded
2. Headache located or transmitted to the Cranium from the Maxillo / Mandibulofacial region, pharynx, paranasal sinuses, neck and ear were excluded.
3. Patients with incomplete clinical profiles, diagnostic & treatment records were excluded.
4. Patients who did not complete the one year follow up were excluded.

Evaluation of Results

The clinical presentations, radiological features, laboratory results, neurophysiological

patterns, treatment and its response on follow up, the progress and complications were documented and tabulated in a Master Chart (Appendix-2). The various parameters of the patients were compared, classified and analysed with specific reference to national and international studies.

Results

A total of 535 patients registered at the Headache Clinic, Institute of Neurology, Madras Medical College & GHH, Chennai between 1st March 2007 and 28th February 2008 were included in the study. Of the total of 535 patients, 402 (75.14%) patients had migraine, 35 (6.54%) had tension type headache, 11 (2.06%) Trigeminal autonomic cephalagia, 9 (1.68%) had other types of primary headaches and 78(14.58%) had mixed tension vascular type of headache.

TABLE 0.1

No	Type of Primary Headache	No of Patients
1	Migraine	402
2	Tension Type Headache	35
3	Trigeminal autonomic cephalalgia	11
4	Other Primary headaches	9
5	Mixed Tension Vascular Headache	78
	TOTAL NO CASES	535

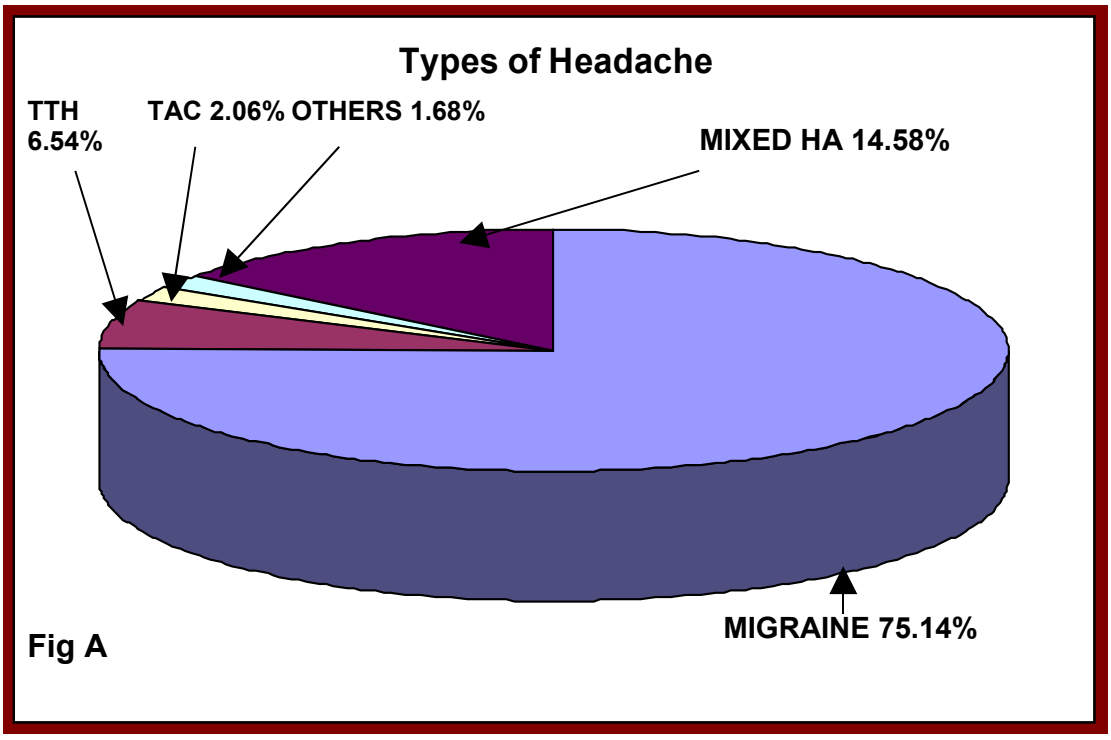


Fig A

I. Classification of Headaches

Of the 402 case of Migraine,192 (47.68%) patients had Migraine without Aura, 38 (9.53%) patients had Migraine with Aura, 92(23.09%) patients had Migraine which presented with & without aura, 70(17.37%) patients had complications of migraine and 10(2.48%) patients had probable migraine. Our study did not include patients with Childhood Periodic Syndromes & Retinal Migraine. The 92 patients who had Migraine which presented with & without aura were considered as a separate group.

TABLE 1.2

No	Type of Migraine	IHS Code	No of Patients
1	Migraine without Aura	1.1	192
2	Migraine with Aura	1.2	38
3	Migraine with and without aura	1.1 / 1.2	92
4	Childhood Periodic Syndromes	1.3	0
5	Retinal Migraine	1.4	0
6	Complications of Migraine	1.5	70
7	Probable Migraine	1.6	10
	TOTAL		402

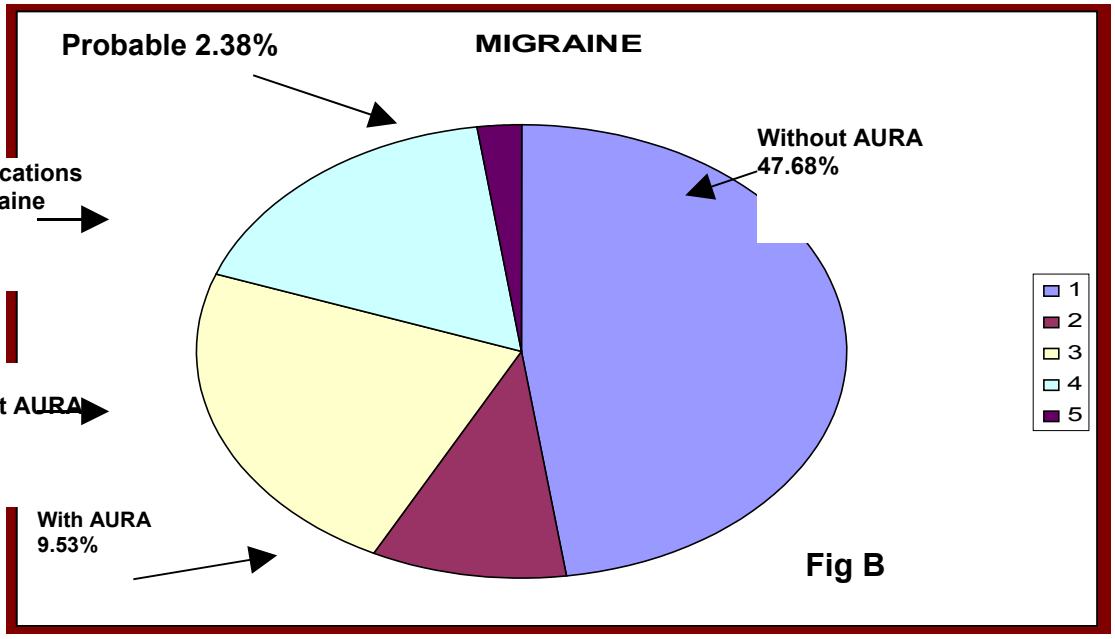
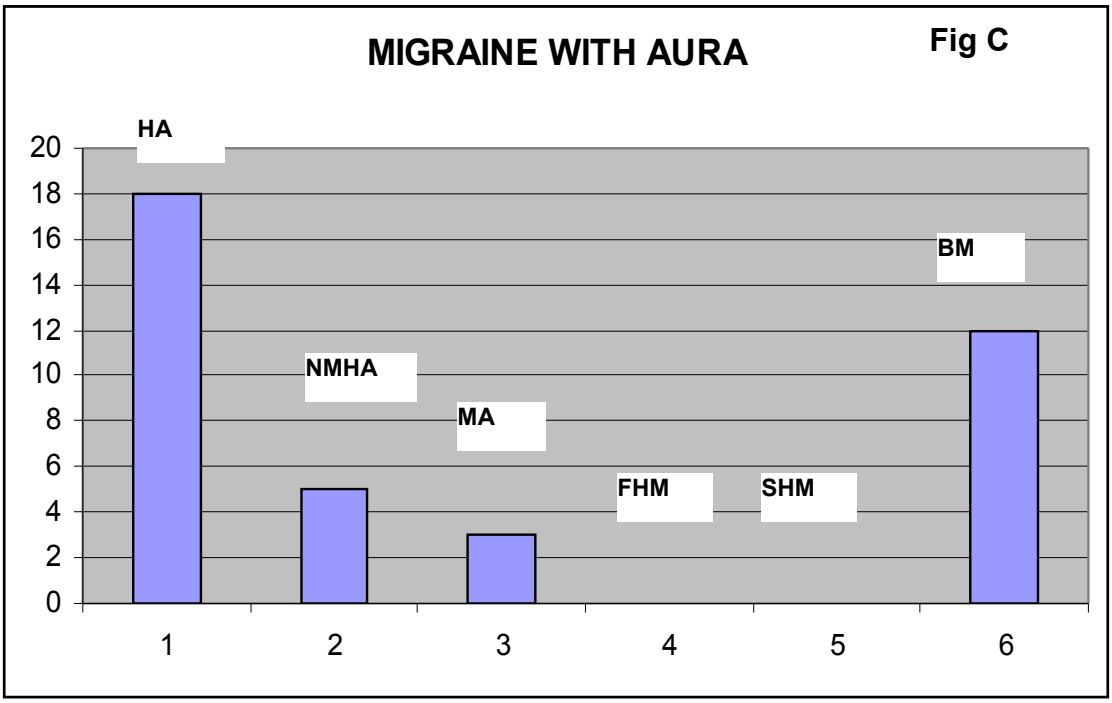


Fig B

Of the patients Migraine with Aura (38 patients) the predominant subtype was Typical Aura with Headache (18 patients). Our study also included 5 patients of Typical Aura with Non Migraine HA, 3 patients with Typical Aura without Headache and 12 patients with Basilar Type Migraine. Our study group did not include patients with Familial Hemiplegic Migraine and Sporadic Hemiplegic Migraine.

TABLE 1.3	No	Type of Migraine with Aura	IHS Code	No of Patients with Migraine with Aura
	1	Typical Aura with Headache	1.2.1	18
	2	Typical Aura with Non Migraine HA	1.2.2	5
	3	Typical Aura without Headache	1.2.3	3
	4	Familial Hemiplegic Migraine	1.2.4	0
	5	Sporadic Hemiplegic Migraine	1.2.5	0
	6	Basilar Type Migraine	1.2.6	12
		TOTAL		38



Of the 70 patients with complications of migraine, 46 patients had Chronic Migraine (9 patients with Chronic Migraine since Onset & 37 patients with Episodic Headache converting to Migraine), 5 patients had Migrainous Infarction (Cerebellar Infarction -3 & Right Occipital Infarction -2) and 19 patients had Migraine Trigerred Seizures (Migraine Terminating as Seizures – 14, Migralepsy - 5). No patients with Status Migrainous or Persistent Aura without Infarction were included in the study.

TABLE 1.4	No	Type of Complications of Migraine	IHS Code	Sub Types	No of Patients	Total No of Patients
	1	Chronic Migraine	1.5.1	Chronic Migraine since Onset	9	46
				Episodic Headache converting to Migraine	37	
	2	Status Migrainosus	1.5.2			0
	3	Persistent Aura without Infarction	1.5.3			0
	4	Migrainous Infarction	1.5.4	Cerebellar Infarction	3	5
				Right Occipital Infarction	2	
	5	Migraine Triggered Seizures	1.5.5	Migraine Terminating as Seizures	14	19
				Migralepsy	5	
		TOTAL				70

In our study, we came across 61 patients with Migraine & Seizures, 19 patients had Migraine Triggered Seizures, 20 patients had Post ictal migrainous headache and 22 patients had Migraine & Coexistent Seizure. Of the 19 patients who had migraine triggered seizures 14 patients had migraine terminating as seizures and 5 patients had Migralepsy.

TABLE 1.5	No	Migraine & Seizures	No of Patients
	1	Migraine Triggered Seizures	19
	2	Post Ictal Migrainous Headache	20
	3	Migraine & Coexistent Seizure	22

Our study also included 5 patients with Ophthalmoplegic Migraine of which 4 had Abducent Nerve palsy and 1 patient had Oculomotor Nerve palsy.

TABLE 1.6	No	Migrainous Ophthalmoplegia	No of Patients
	1	Abducent Nerve Palsy	4
	2	Oculomotor Nerve Palsy	1

Out of a total of 535 patients registered at the Headache Clinic, 35 (6.54%) had tension type headache, of which 5 patients had Infrequent Episodic TTH, 7 patients had Frequent Episodic TTH, 19 patients had Chronic TTH, and 4 patients had Probable TTH.

TABLE 1.7	No	Type of TTH	IHS Code	No of Patients
	1	Infrequent Episodic TTH	2.1	5
	2	Frequent Episodic TTH	2.2	7
	3	Chronic TTH	2.3	19
	4	Probable TTH	2.4	4
		TOTAL		35

Out of a total of 535 patients registered at the Headache Clinic 11 (2.06%) had Trigeminal autonomic cephalagia of which 5 patients had Cluster Headache, 4 patients had Paroxysmal hemicrania, 2 patients had Short Lasting Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing. No case of Probable TAC was registered.

TABLE 1.8

No	Type of Trigeminal Autonomic Cephalalgia	IHS Code	No of Patients
1	Cluster Headache	3.1	5
2	Paroxysmal hemicrania	3.2	4
3	SUNCT	3.3	2
4	Probable TAC	3.4	0
	TOTAL		11

Of the 5 patients who had Cluster Headache 3 were classified as Episodic Cluster Headache and 2 as Chronic Cluster Headache. Of the 4 patients who had Paroxysmal Hemicrania, 2 were classified as Episodic Paroxysmal Hemicrania and 2 as Chronic Paroxysmal Hemicrania.

TABLE 1.9

No	Type of Cluster Headache	IHS Code	No of Patients
1	Episodic Cluster Headache	3.1.1	3
2	Chronic Cluster Headache	3.1.2	2
	TOTAL		5

TABLE 1.10

No	Type of TAC	IHS Code	No of Patients
1	Episodic Paroxysmal Hemicrania	3.2.1	2
2	Chronic Paroxysmal Hemicrania	3.2.2	2
	TOTAL		4

Primary headaches were few among the registered patients at the Headache Clinic. 9(1.68%) patients had primary headaches, of which 2 patients had Primary Stabbing Headache, 1 patient had Primary Headache associated with Sexual Activity, 1 patient had Primary hypnic Headache, and 5 patients had New Daily Persistent Headache.

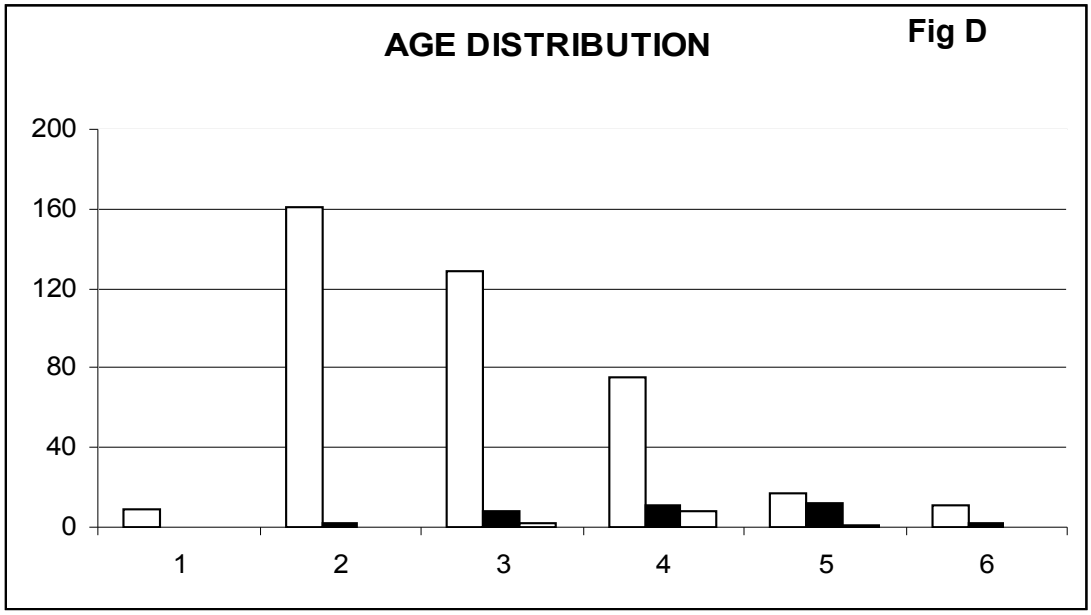
TABLE 1.11	No	Other Primary Headaches	IHS Code	No of Patients
	1	Primary Stabbing Headache	4.1	2
	2	Primary Cough Headache	4.2	0
	3	Primary Exertional Headache	4.3	0
	4	Primary Headache ass with Sexual Activity	4.4	1
	5	Primary Hypnic Headache	4.5	1
	6	Primary Thunderclap Headache	4.6	0
	7	Hemicrania Continuua	4.7	0
	8	New Daily Persistent Headache	4.8	5
		TOTAL		9

2. Epidemiological Data

The patients were analysed for [1] **Age Distribution:**

TABLE 2.12	No	Age Group	No of Patients with Migraine	No of Patients with TTH	No of Patients with Mixed Tension Vascular Headache	No of Patients with TAC
	1	0-10yrs	9	0	0	0
	2	11-20yrs	161	2	0	0
	3	21-30yrs	129	8	0	2
	4	31-40yrs	75	11	41	8
	5	41-50yrs	17	12	29	1
	6	51 + yrs	11	2	8	0

Migraine was most common in the 2nd and 3rd decades , 290 out of 402 patients were between 11-30years of age (11-20yrs - 161 patients, 21-30yrs – 129 patients, 31-40yrs – 75patients). Majority of the patients (23 out of 35 patients) with Tension type headache were in the 4th and 5th decade of life (21-30yrs - 8 patients, 31-40yrs – 11 patients, 41-50yrs – 12patients). Tension vascular type of headache matched the tension type headache in age distribution, being more common in 4th and 5th decade of life. Trigeminal Autonomic Cephalalgia was more common between 31-40years of age - 8 patients.



In patients with migraine triggered seizures of the total 19, 10 patients were in the age group of 11-20years. Basilar migraine was also found to be common in the age group of 11-20years(9 out of 12 patients). Patients with migrainous stroke were between 21-40years of age and Chronic migraine was noticed in 4th and 5th decade of life predominantly.

TABLE 2.13

No	Age Group	No of Patients
1	0-10yrs	1
2	11-20yrs	10
3	21-30yrs	5
4	31-40yrs	3
5	41-50yrs	0
6	51 + yrs	0

[2] Gender Distribution: The patients analysed for gender distribution. Female patients dominated in the category of migraine 299 of the 402 patients, in tension headache 26 of the 35 patients and in tension vascular headache 57 of 78 patients, while in trigeminal autonomic cephalalgia 9 of the 11 patients were male.

TAB 2.14	No	Sex Distribution	Migraine	TTH	Mixed TVH	TAC
	1	Male	103	9	21	9
	2	Female	299	26	57	2

Complications of migraine was more common in female patients as evidenced by all the five patients with migrainous stroke were females. Out of the 19 patients with migraine triggered seizures 12 were female and 7 were male. Chronic migraine was predominantly seen in females (30 out of 46 patients).

[3] Family Predilection: A detailed clinical history as recorded with the proforma in appendix -1 was analysed for the various clinical parameters in each case. Family history was positive in 229 patients (56.96%) with migraine, 11 patients (31.43%) with tension headache, 51 patients (65.38%) with tension vascular headache and none with TAC.

TAB 2.15	No	Family History	Migraine	TTH	Mixed TVH	TAC
	1	Positive F.H	229	11	51	0
	2	Negative. F.H	173	24	27	11

3. Clinical Presentations

A] Presentation:

1. Migraine: Headache in patients with migraine, 313 presented with unilateral headache, of which 208 patients experienced shift of sides while 105 patients had always a unilateral headache. Most of the episodes of migrainous headache in our patients lasted more than 12 hours [141 patients]. The migrainous headaches were predominantly temporal (233 patients). Most of the patients experienced a throbbing type of headache (271 patients).

TABLE 3.16	No	Duration	Migraine	TTH	Mixed TVH	TAC
	1	1-6 hours	56	5	0	9
	2	6-12 hours	92	10	9	2
	3	12-24hours	141	18	35	-
	4	More than 24 hours	113	2	34	-

TABLE 3.17	No	Location	Migraine	TTH	TAC
	1	Unilateral	313	4	9
		Unilateral with shifting sides	208	-	-
		Same Unilateral side	105	-	-
	2	Bilateral	89	31	2

2. Tension Type Headache: In patients with TTH 18 presented with headache of more than 12hours duration, while 10 patients had duration of 6-12 hours and 5 patients had duration of 1-6hours. The headache was holocranial in 31 of the 35 patients while it was hemicranial in the other 4 patients. 31 of these patients who experienced bilateral headaches had headaches confined to the frontal region. Most of the patients experienced aching type of headache (23 patients), while it was band like in 11 patients and throbbing in 6 patients. In most of these patients the headache radiated to nuchal region and there was an associated scalp tenderness during the episodes of headache.

3. Tension Vascular Headache: Of the 78 patients diagnosed most patients had a duration of more than 12 hours(69 patients). 62 patients had a earlier diagnosis of Migraine without Aura whose frequency of episodes increased over a period of few years to chronic migraine / transformed migraine (more than 15 episodes per month) which progressed to bilateral holocranial headache. The character of headache changed from the typical throbbing nature to diffuse aching pain at times band like pain with radiation to the neck. As the headache gets transformed patients experience fewer associated symptoms of migraine.

4. Trigeminal Autonomic Cephalalgia: In patients with Trigeminal Autonomic Cephalagia 9 presented with headache of less than 6 hours duration, while 2 patients had a duration of 6-12 hours. 2 of the patients experienced bilateral headaches while 9 had unilateral headaches with most (9 patients) confined to the frontal temporal region and was aching in character.

TABLE 3.18	No	Location	Migraine	TTH	TAC
	1	Frontal	95	19	0
	2	Temporal	233	8	0
	3	Occipital	47	5	0
	4	Parietal	27	3	2
	5	Fronto Temporal	0	0	9

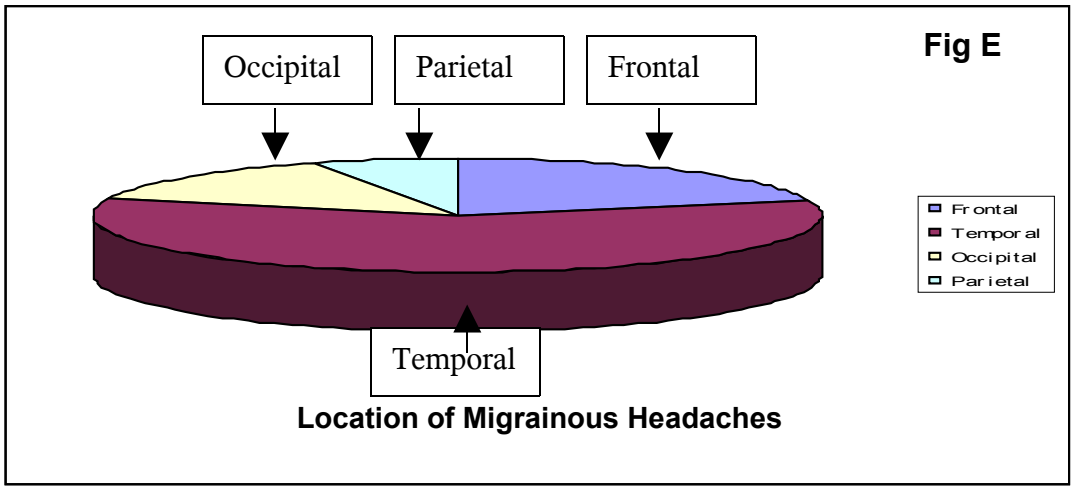


Fig E

Location of Migrainous Headaches

TABLE 3.19

No	Type of Headache	Migraine	TTH	Mixed TVH	TAC
1	Throbbing	271	6	12	2
2	Pricking	56	3	1	1
3	Aching	47	23	16	8
4	Band Like	0	11	24	0
5	Diffuse Dull Ache	13	0	25	0
6	Burning	15	0	0	0

B] Symptoms, Aggravating and Relieving Factors:

1. Migraine: The predominant premonitary symptom was yawning (98patients), followed by fatigue, irritability, sense of feeling low and over eating in few patients. Associated symptoms in order occurrence being photophobia, phonophobia, blurring of vision, nausea, vomiting and giddiness. Loss of consciousness was reported in 49 patients and ipsilateral pain in limbs in 45 patients. Autonomic symptoms like lacrimation, redness and transient syncope were reported in few patients. The most common aggravating factor in our study group was mental stress, while physical stress, head bath, bright sunlight, lack of sleep, travel and consumption of chocolates were also commonly reported. Head bath as an aggravating factor has been observed in 267 patients. The relieving factors were mostly rest and analgesics or topical applications and sleep.

TABLE 3.20	No	Premonitary Symptoms	Migraine	TTH	TAC
	1	Yawning	98	1	-
	2	Fatigue	57	14	-
	3	Irritability	39	19	4
	4	Feeling low	20	1	-
	5	Over Eating	22	0	-

2. Tension Type Headache: The predominant premonitary symptom was feeling low. Associated symptoms being phonophobia (27 patients) or photophobia, nausea or vomiting and in few patients paresthesia and giddiness. The most common aggravating factor in our study group was mental stress, while physical stress, travel, head bath and lack of sleep were also commonly reported. The relieving factors were mostly rest and analgesics or topical applications, pressure and coffee / tea ingestion.

TABLE 3.21	**No**	**Associated Symptoms**	**Migraine**	**TTH**	**MixedTVH**	**TAC**
	1	Blurring of Vision	372	0	5	4
	2	Photophobia	395	8	33	9
	3	Phonophobia	398	27	12	9
	4	Nausea	373	19	42	10
	5	Vomiting	128	5	10	10
	6	Giddiness	148	7	3	0
	7	Lacrimation	27	1	0	11
	8	Paresthesia	34	10	9	0
	9	Tremulousness	38	0	0	0
	10	Transient LOC	49	0	0	0
	11	Confusion	19	0	0	0
	12	Redness of Eyes	16	0	0	11
	13	Pain ipsilateral limbs	45	0	0	0
	14	Drooping of eyelids	0	0	0	7
	15	Nasal Stuffiness	0	0	0	9

3. Tension Vascular Headache: The predominant associated symptoms were nausea (42 patients), with photophobia (33 patients), phonophobia (12 patients), paraesthesia (9 patients), giddiness (3 patients) in order of occurrence. The most common aggravating factor in our study group was mental stress, while physical stress, travel, head bath and lack of sleep were also commonly reported. The relieving factors were mostly analgesics, rest, sleep, pressure and coffee / tea ingestion. Of these 3 patients who were on multiple analgesics prescribed, responded on withdrawal of drugs.

4. Trigeminal Autonomic Cephalagia: The predominant associated symptom was nausea and

vomiting (10 patients), with redness of eyes, nasal stuffiness, phonophobia, photophobia, drooping of eyelids and blurring of vision the other common associated symptoms in order of occurence. Aggravating factors were not reported in most patients in this study though alcohol intake, sunlight and lack of sleep were reported. The relieving factors were mostly analgesics or topical applications, pressure and coffee / tea ingestion.

TABLE 3.22

No	Aggravating Factors	Migraine	TTH	MixedTVH	TAC
1	Head Bath	267	15	18	0
2	Physical Stress	281	22	21	0
3	Mental Stress	367	25	47	0
4	Sunlight	241	12	31	1
5	Travel	193	18	11	0
6	Lack of Sleep	235	19	22	2
7	Perfumes	53	0	0	0
8	Petrol/Diesel Smell	22	0	0	0
9	Concentrated Reading	44	4	8	0
10	Chocolates	192	0	18	0
11	Cold Food/Drink	176	0	10	0
12	Close Rooms - Theatre/Auditorium	48	8	18	0
13	Alcohol Intake	0	0	0	3

TABLE 3.23

No	Releiving Factors	Migraine	TTH	Mixed TVH	TAC
1	Rest & Analgesics	398	37	26	0
2	Rest & Topical Applications	112	14	4	8
3	Sleep	356	11	14	1
4	Rest	65	6	9	1
6	Local Pressure	0	26	5	6
7	Coffee / Tea	2	16	7	1
8	Analgesic Abstinence	0	0	3	0

C] Aura:

The preponderant type of aura reported in our migranous patients was visual aura in 113 patients while sensory aura was reported in 32 patients and both in 9 patients. The visual aura was predominantly in the form of flickering of lights in 67 patients, while zig-zag lines, scintillating scotomas and fortification spectra was noted in 22 patients, 16 patients and 8 patients respectively. The sensory aura seen was commonly in the form of paresthesia (27 patients). In patients with migraine triggered seizures of the total 19 patients, 14 patients had visual aura.

TABLE 3.24	No	Aura	No of Patients	Type of Aura	
	1	Visual Aura	113	Fortification Spectra	8
				Scintillating Scotoma	16
				Flickering Lights	67
				Zig Zag Lines	22
	2	Sensory Aura	32	Paresthesia	27
				Numbness	5
	3	Visual & Sensory aura	9		

D] Migraine Triggered Seizures:

Of the 19 patients who had Migraine Triggered Seizures 14 patients had Migraine Terminating as Seizures and 5 patients had Migralepsy. On analysing the seizure pattern of these 19 patients, 9 patients had GTCS, 5 patients had CPS and 5 patients presented with prolonged LOC of more tha 20 minutes, suggesting a generalised seizure rather than an associated autonomic dysfunction.

TABLE 3.25	No	Migraine Triggered Seizures	No of Patients
	1	GTCS	9
	2	CPS	5
	3	Prolonged LOC	5

TABLE 3.26	No	M T Seizures & Aura	No of Patients
	1	Without Aura	5
	2	With Visual Aura*	14

*Aura was colourless, unformed flickering of light and scintillating scotomas of longer duration

TABLE 3.27	No	Time between Migraine & onset of seizure	No of Patients
	1	2-3hrs	5
	2	3-6 hrs	8
	3	6-12 hrs	4
	4	>12 hrs	2

E. Associated Conditions:

1. Migraine: In our study of 402 patients with migraine the following clinical conditions were seen associated – namely Healed granulomatous lesions on CT, Seizures, Hypertension, Head Injury, Sinusitis, Psychiatric Changes and Stroke

2. Tension Type Headache: In our study of 35 patients with TTH the following clinical conditions were seen associated – namely healed granulomatous lesions in CT, Hypertension and Head Injury.

3. Tension Vascular Headache: In our study of 78 patients with TVH, 15 patients had associated mental depression.

4. Trigeminal Autonomic Cephalagia: In our study of 11 patients with TAC, no significant clinical

conditions were associated.

TABLE 3.28	No	Clinical Diagnosis	Migraine	TTH	MIxedTVH	TAC
	1	Seizures	61	0	0	0
	2	Granulomatous Diseses	72	12	0	0
	3	Stroke	9	0	0	0
	4	Head Injury	23	12	0	0
	5	Hypertension	32	15	0	0
	6	Sinusitis	23	0	0	0
	7	Transient Global Amnesia	2	0	0	0
	8	Psychiatric Changes	10	3	15	0

4. Diagnostic Tests

1. Electrophysiology:

Migraine: EEG was taken in 54 of the 402 patients with migraine. 28 of the 54 patients showed non specific slowing in posterior region, while 5 patients showed spikes and sharp waves in occipital region. Spikes not altered by eye opening. 21 of the 54 patients had no specific changes.

(b) Migraine Triggered Seizures: EEG was taken in all the 19 patients with Migraine Triggered Seizures. Changes were seen in 10 out of 19 pts, 6 patients had spikes in posterior head region. Spikes and sharp waves during visual aura showed waxing and waning separated by completely normal EEG activity despite the persistence of symptomsand these spikes were not altered by eye opening. 4 pts had non specific slowing.

(c) Tension Type Headache: EEG was taken in 12 of the 35 patients with TTH, 2 of these patients showed non specific slowing in posterior region, while 10 of the patients had no specific changes

(d) Trigeminal Autonomic Cephalagia: EEG was taken in 4 of the 11 patients with TAC and none showed specific changes.

TABLE 4.29	No	Migraine -EEG	Migraine	TTH
	1	No Changes	21	10
	2	Non Specific Slowing in Posterior Region	28	2
	3	Spikes & Sharp Waves in Occipital Region	5	0

2. Autonomic Function Tests: A case control age matched study of heart rate variability was done in 17 patients with migraine. The values as depicted in tables 4.30 and 4.31 for controls and patients respectively represent (1) Heart Rate /minute in resting state (2) Resting Systolic BP (3) Resting Diastolic BP (4) LF - Low Frequency Heart Rate Variation (5) High Frequency Heart Rate Variation (6) LF/HF ratio (7) Mean Heart Rate (8) Mean RR Interval.

Low Frequency Heart Rate Variation (LF): A mean control value of 40.34 and a mean test value for migraineurs of 37.4 with a SD (5.83) p value of 0.2837. Hence concluded that there is no significance.

High Frequency Heart Rate Variation (HF): A mean control value of 59.93 and a mean test value for migraineurs of 62.60 with a SD (11.18), p value of 0.620. Hence concluded that there is no significance.

LF/ HF Ratio: A mean control value of 0.706 and a mean test value for migraineurs of 0.709 with a SD (0.411), p value of 0.975. Hence concluded that there is no significance.

Mean Heart Rate: A mean control value of 74.0 and a mean test value for migraineurs of 76.20 with a SD (8.64), p value of 0.0327. Hence concluded that this value shows significant difference between the control and test.

Mean RR Interval: A mean control value of 0.813 and a mean test value for migraineurs of 0.842 with a SD (0.124), p value of 0.738. Hence concluded that there is no significance.

TABLE 4.30

CONTROL										
S.NO.	AGE	GENDER	HR-/min RESTING	SBP- mmHg RESTING	DBP- mmHg RESTING	LF	HF	LF/HF	MEAN HR	MEAN RR
C1	30	F	73	116	76	38.60	61.40	0.630	79.00	0.76
C2	42	F	81	114	68	35.70	64.30	0.560	66.00	0.91
C3	18	F	68	124	76	38.80	61.20	0.640	62.00	0.96
C4	25	F	79	110	70	35.50	64.50	0.550	66.00	0.91
C5	21	F	80	124	76	38.50	61.50	0.630	79.00	0.75
C6	15	F	78	120	80	36.20	68.30	0.682	74.00	0.81
C7	43	F	79	122	78	38.30	61.70	0.620	62.00	0.95
C8	27	F	81	124	84	35.40	64.60	0.560	70.00	0.91
C9	35	F	81	124	80	42.30	57.70	0.750	69.00	0.75
C10	19	F	78	120	76	35.80	64.20	0.560	74.00	0.81
C11	27	F	79	118	60	41.80	58.20	0.720	69.00	0.88
C12	19	F	80	120	78	38.70	61.30	0.630	62.00	0.96
C13	40	M	74	116	78	42.30	57.70	0.733	79.00	0.75
C14	25	M	82	120	84	54.90	45.10	1.220	70.00	0.89
C15	47	M	82	120	80	54.80	45.20	1.210	62.00	0.89
C16	19	M	81	124	80	42.30	57.70	0.750	69.00	0.75
C17	20	F	78	120	76	35.80	64.20	0.560	74.00	0.81
			78.47059	119.7647	76.47059	40.33529	59.92941	0.706176	69.76471	0.85370

TABLE 4.31

PATIENTS WITH MIGRAINE										
S.NO.	AGE	GENDER	HR-/min RESTING	SBP -mmHg RESTING	DBP -mmHg RESTING	LF	HF	LF/HF	MEAN HR	MEAN RR
P1	32	F	62	110	78	36.50	63.50	0.575	57.83	1.224
P2	45	F	95	130	90	27.90	72.10	0.387	68.33	1.130
P3	16	F	80	120	80	43.80	56.20	0.779	84.00	0.715
P4	23	M	85	120	70	15.70	84.30	0.186	73.46	0.839
P5	21	F	96	120	70	44.80	55.20	0.812	92.82	0.652
P6	13	M	72	90	70	20.60	79.40	0.259	71.01	0.859
P7	41	F	68	108	70	17.50	82.50	0.212	63.23	0.951
P8	24	F	65	100	70	39.60	60.40	0.656	75.31	0.901
P9	34	F	78	100	70	39.60	60.40	0.656	83.16	0.723
P10	16	M	80	100	70	37.90	62.10	0.610	79.51	0.759
P11	27	F	85	110	70	36.90	63.10	0.585	84.04	0.716
P12	18	F	70	90	60	17.70	82.30	0.215	68.37	0.880
P13	41	F	80	120	70	34.70	65.30	0.531	78.67	0.763
P14	24	F	92	120	80	72.00	28.00	2.571	91.72	0.655
P15	48	F	92	120	80	51.00	49.00	1.041	83.65	0.720
P16	17	F	72	90	60	49.40	50.60	0.976	66.08	0.980
P17	21	M	74	140	80	50.20	49.80	1.008	71.12	0.846
			79.17647	111.0588	72.82353	37.40	62.60	0.709	76.02	0.842

3. CT Scan:

(a) Migraine: CT scan of Brain was taken in all of the 402 patients, of whom 83 had changes. The most common change reported in CT scan brain was calcified granulomas in 69 patients, gliosis in 8 patients and basal ganglia calcification in 6 patients. CT scan of the para-nasal sinuses was performed in 53 patients of which 18 showed maxillary sinusitis while it was normal in 35 patients. CT scan Brain was taken in all the 19 patients with Migraine trigerred seizures, was found to be normal in all patients.

TABLE 4.32

No	Migraine Neuro Imaging - CT Brain	No of Patients
1	Normal	319
2	Abnormal	83
	Total	402

TABLE 4.33

No	CT Brain In Migraine	No of Patients
1	Calcified Granuloma	69
2	Gliosis	8
3	Basal Ganglia Calcification	6
	TOTAL	83

TABLE 4.34

No	CT Paranasal Sinues in Migraine	No of Patients
1	Normal	35
2	Chronic Maxillary Sinusitis	18
	TOTAL	53

(b) Tension Type Headache: CT scan of brain was taken in all of the 35 patients, of whom 15 had changes. The most common change reported in CT scan brain was calcified granulomas in 12 patients and gliosis in 3 patients..

TABLE 4.35

No	CT Brain in TTH	No of Patients
1	Normal	20
2	Calcified Granuloma	12
3	Gliosis	3

(c) Trigeminal Autonomic Cephalagia: CT scan of brain was taken in all of the 11 patients with none showing abnormality.

5. Prophylaxis

1. Migraine: The patients with migraine were given prophylactic therapy with either Propronolal (20-160mg), or Amitryptylline(12.5-50mg), or Propronolal (20-160mg) and Amitryptylline (12.5-50mg), or Flunarizine(5mg), or Topiramate(50mg), or Sodium Valproate ER(500mg).

TABLE 5.36

No	Drugs	Patients
1	Propronolal(20-160mg)	99
2	Amitryptylline(12.5-50mg)	51
3	Propronolal (20-160mg)+ Amitryptylline (12.5-50mg)	215
4	Flunarizine(5mg)	27
5	Topiramate(50mg)	5
6	Sodium Valproate ER(500mg)	5
	TOTAL	402

TABLE 5.37

No	Migraine	No of Patients	Disease Free period			Decreased Intensity & Frequency of Pain	No Response
			>1 Year	6Months-1year	Less than 6months		
1	Propronolal(20-160mg)	99	15	42	18	24	-
2	Amitryptylline(12.5-50mg)	51	9	13	10	13	6
3	Propronolal (20-160mg) + Amitryptylline (12.5-50mg)	215	37	66	68	44	-
4	Flunarizine(5mg)	27	9	8	6	4	-
5	Topiramate(50mg)	5	-	-	3	2	-
6	Sodium Valproate ER(500mg)	5	-	2	-	2	1

2. Migraine Triggered Seizures: All patients were treated with anti migrainous prophylaxis with T.Propronolal (40-120mg) or T.Flunarizine (5-10mg) and analgesics during episodes of headache. T. Sodium valproate (600mg) was given to patients who had seizures on follow up. Follow up period was 1 year. Of the 19 patients 16 patients had 1 year incident free period, while 3 patients had seizure attacks in between which were treated with Sodium valproate (600mg).

3. Tension Type Headache: All patients were treated with Amitryptiline (10-50mgms). Of the 35 patients 3 patients responded with a disease free interval of 1year, 9 patients with a disease free interval of 6 months to 1 year, 9 patients with a disease free interval of less than 6months, 11 patients

responded with decreased intensity and frequency of episodes while 3 patients did not show any response.

3. Tension Vascular Headache: All 78 patients were treated with Amitryptiline (10-50mgms) and Propronolol (40-160mgms). Of the 78 patients 63 patients responded with decreased intensity and frequency of episodes while 15 patients did not show any response.

4. Trigeminal Autonomic Cephalagia: All patients who presented with acute pain were treated with analgesics (Naproxen 50mgms, Ibuprofen 400mgms, indomethacin 25-75mgms, or occasionally steroids). Prophylaxis was provided for 11patients of which 8 were given Amitryptiline (25-50mgms), 3 patients were given Propranolol (80-120mgms). Of the 8 patients given Amitryptiline 7 patients responded with a disease free interval of 1year, 1 patient with a disease free interval of 6 months to 1 year. Of the 3 patients given Propranolol all 3 patients responded with a disease free interval of 1year.

Discussion

This study at the Headache Clinic of the Institute of Neurology, Madras Medical College & GGH, Chennai between 1st March 2007 and 28th February 2008, encompassed a period of one year.

I DEMOGRAPHY OF PRIMARY HEADACHES:

I. INCIDENCE: Of the 535 patients of primary headaches included in this study 402 (75.14%) patients had migraine, 35 (6.54%) had tension type headache, 11 (2.06%) had Trigeminal autonomic cephalalgia, 9 (1.68%) had other types of primary headaches and 78(14.58%) had mixed tension vascular type of headache. This profile of headaches in our study was is in inverse correlation with many international studies. [Lipton et al 2002].[83] Tension type headaches are considered the most common form of headache in the general population with a prevalance of nearly 80% while the prevalance of migraine is pegged at 16% in various international studies. In contrast, migraine is a more common form of headache reported in clinical practice. This variance is attributed to self treatment of tension type headaches by the general population. This variation reported in our study correlates with the study of Lance and Curran [1964] [84]

In our study, 78 patients were observed to present with features of tension headache and later had features of migraine. This substantial group of patients, who conformed to the diagnostic criteria of more than one type of headache have been grouped under a common

head “mixed or overlap headaches” - tension vascular headache. This observation of ours is in consonance with the observations of Lance and Curran [1964].[84]

A. Incidence of Migraine: In our study of the 402 cases of Migraine, 192 (47.68%) patients had Migraine without Aura, 38 (9.53%) patients had Migraine with Aura, 92(23.09%) patients had Migraine which presented with and without aura, 70(17.37%) patients had complications of migraine and 10(2.48%) patients had probable migraine. The ratio of Migraine without Aura and Migraine with Aura in our study is calculated approximately at 5:1 which correlates with international studies [Allan H Ropper 2005].[85].However the ratio narrows down to 1.5:1 if migraine without aura is compared against migraine with and without aura. Our study did not include patients with Childhood Periodic Syndromes & Retinal Migraine. This is attributable to the existence of independent Institutes for Children and Eye Diseases within close reach of our institute. In our study of the 402 cases of Migraine, 92 (23.09%) patients had Migraine which presented with or without aura. These 92 patients were placed as a separate group. This group of patients have has been identified and given particular mention in the ICHD 2004 classification. “*Many patients who have frequent attacks with aura also have attacks without aura (code as 1.2 Migraine with aura and 1.1 Migraine without aura).”* [86]. Yet, the world literature lacks specific references of this group of cases, their epidemiological significance, clinical behaviour and therapeutic importance. Similarly the ICHD-2 lacks any specific guidelines about the statistical placement of this group of cases. Hence, in our study we have placed this group of patients as a separate sub entity within the entity migraine. A detailed study of the epidemiology significance, clinical and therapeutic behaviour of these patients is needed.

B. Incidence of Tension Type Headaches: In this study of 535 headache patients 35 (6.54%) had tension type headache, of which 5 patients had Infrequent Episodic TTH, 7 patients had Frequent Episodic TTH, 19 patients had Chronic TTH, and 4 patients had

Probable TTH. The prevalence of Tension type headache was 40.5% in epidemiological studies by Schwartz et al (1998)[87]. This study also included 3 patients of TTH with chronic drug overuse who have been classified under tension vascular headache.

C. Incidence of Trigeminal Autonomic Cephalagias: Out of a total of 535 patients registered at the Headache Clinic 11 (2.06%) had Trigeminal autonomic cephalalgia of which 5 patients had Cluster Headache, 4 patients had Paroxysmal hemicrania, 2 patients had Short Lasting Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing. No case of Probable TAC was registered.

In this study the incidence of cluster headache is 1% in contrast to the observation made, that cluster headache appears to be less common in India than in the west [Ambar Chakravarthy et al 2004][88]. The prevalence of cluster headache estimated in U.S is around 0.1%.

A total of 535 patients registered at the Headache Clinic 9 (1.68%) had other types of primary headaches of which 2 patients had Primary Stabbing Headache, 1 patient had Primary Headache associated with Sexual Activity, 1 patient had Primary Hypnic Headache, and 5 patients had New Daily Persistent Headache

II. AGE AND GENDER DISTRIBUTION - A. Age Distribution:

(i) Migraine: Of the 402 patients having migraine most of them were between 11-40years of age (11-20yrs - 161 patients, 21-30yrs – 129 patients, 31-40yrs – 75patients).In this study Migraine with aura peaked around 13-15 years of age in males(6 out of 11) and around 22-25 years in females(15 out of 27). The onset and peaking in both male and female patients in this study is 8-10 years delayed in contrast to that observed in western population by Stewart et al [1991]. According to him the incidence of migraine with aura in females peaked between ages 12 and 13 (14.1/1000 person-years); and in males, migraine with aura

peaked in incidence several years earlier, around 5 years of age at 6.6/1000 person-years. Before puberty, migraine prevalence is higher in boys than in girls. The peak incidence of basilar migraine in this study was around 10-15 years which correlates with the international literature. As adolescence approaches, incidence and prevalence increase more rapidly in girls than in boys. The prevalence increases throughout childhood and early adult life until approximately age 40, after which it declines [Stewart WF et al 1991][89]. If the migraine headaches persist beyond 40 years of age there is more chance for transformation into chronic migraine. Transformation is proportional is proportional to the chronicity of headache with early onset in teens.

(ii) Tension Type Headache: Of the 35 patients having Tension type headache most of them were between 31-50years of age (23 out of 35 patients, 31-40yrs – 11 patients, 41-50yrs – 12patients). The average age of onset of TTH is higher than in migraine in this study, namely 25 to 30 years. The prevalence peaks between ages 30 to 39 and decreases slightly with age [Rasmussen BK 1995][90]. Of the 78 patients who had features of mixed tension vascular type of headache 41 patients were between 31-40 years of age.

(iii) Trigeminal Autonomic Cephalagias: Of the 11patients having cluster headache most of them (8 out of 11 patients) were between 31-40years, which correlates with the observation by Bohra et al.2002[91]

B. Gender Distribution: The patients analysed for gender distribution. in tension headache 26 of the 35 patients were female, while in cluster headache 9 of the 11 patients were male

(i) Migraine: Of the 402 patients with migraine 73% of them were females. Female predominance is noted in all groups including migraine with aura, migraine without aura, basilar migraine, migraine triggered seizures, migrainous infarction and chronic migraine. Menstrually related migraine was noticed in 46 patients whereas pure menstrual migraine was present in 3 patients. The American Migraine Study-1 (AMS-1)[10] and AMS-II, collected

information from 15,000 households representative of the US population in 1989 and 1999. Finally, the American Migraine Prevention and Prevalence study (AMPP) replicated, in its first research phase, the methods of AMS-I and AMS-II.12 In these three very large studies, the prevalence of migraine was about 18% in women and 6% in men [Abu-Arefeh.I et al 1994] [92].

(ii) Tension Type Headache: The female-to-male ratio of TTH in this study is 3:1, again showing a female preponderance. In western countries the ratio is 5:4 indicating that, unlike migraine, women are affected only slightly more than men Stovner L,et al 2007[93]. Of the 78 patients with Tension Vascular Headche 57 were female.

(iii) Trigeminal Autonomic Cephalagias: In this study male predominance is noted in concordance with the observation by Manzoni GC [Manzoni GC et al 1998][94]. Of the two female patients included under TAC, one had SUNCT and other had episodic cluster headache**.** .

C. Family Predilection: Family history was positive in 229 patients (56.96%) with migraine. Of the 38 patients with migraine with aura 25 out of 38 patients had a first degree relative suffering from headache. Positive family history is also noticed in patients with basilar migraine and in patients with migrainous strokes. Russell MB et al [1995] has stated that first degree relatives of patients with migraine with aura had a three –four fold increased risk of migraine and it is two fold in first degree relatives patients with migraine with out aura[95]. 11 patients (31.43%) with tension headache in this study had a positive family history. Family background of some form of headache in 40% of patients with Tension Type Headache has been reported by Freidman A et al [1964][96], while Russell MB et al [1999][97] reported that parents siblings and children had a 2.1 to 3.9 fold increase risk of chronic TTH during their life time. 51 patients (65.38%) with tension vascular headache had a family history of migraine in 40 patients and of TTH in 11 patients. Our study group doesn't have a family history in the

TAC group even though Kudrow L et al.[1980][98] in his study has reported a high risk of cluster headache by 14 times among first degree relatives of cluster headache patients

II. CLINICAL PRESENTATIONS OF PRIMARY HEADACHES

A. MIGRAINE:

i) Location and Character: In patients with migraine 313 presented with unilateral headache, with 208 of these patients experiencing shift of sides. The headache was predominantly temporal (233 patients). Most of the patients experienced a throbbing type of headache (271 patients) with a maximal duration of 12-24 hours in 141 patients. The predominant premonitory symptom was yawning (98patients), with nausea, phonophobia, photophobia and blurring of vision the commonest associated symptom in order of occurrence. Loss of consciousness was reported in 49 patients and ipsilateral pain in limbs in 45 patients more commonly in upper limbs and it represents extension of allodynia. Some authors consider it as an aura as it precedes the headache [Guiloff RJ 1988][99].

ii] Aura: Among the several types of aura, visual aura was more common (113 of 130 cases) which is in concordance with the literature. Negative visual aura was more common than positive symptoms and often occurred in isolation.(Kelman L et al 2004)[100] Typical visual aura but headache not fulfilling the criteria for migraine was seen in 5 patients. Three patients while on treatment for migraine headaches with aura, later had only aura alone without headache. The visual aura was predominantly in the form of flickering of lights in 67 patients, while zig-zag lines, scintillating scotomas and fortification spectra was noted in 22 patients, 16 patients and 8 patients respectively. This is in correlation with most international studies. When patients with typical aura with migraine headache become older, their

headache may lose migraine characteristics or disappear completely even though auras continue. Sensory aura was reported in 32 patients and both in 9 patients. The sensory aura seen was commonly in the form of paraesthesia (27 patients). [Christopher J Boes et al 2004] [101]

iii] Aggravating and Relieving Factors: In our study the most common aggravating factors were mental stress, while physical stress and lack of sleep were also commonly reported. Head bath as an aggravating factor has been observed in 267 patients. A similar observation has been referred to by Ravishankar et al 2006[9]. Common triggers of migraine are well established but 'hair wash or head bath' as a trigger for migraine has so far not been reported. This prospective study analysed this unusual trigger link in 94 out of 1000 Indian patients who fulfilled the International Headache Society criteria for migraine. In 11 patients, hair wash was the only trigger; in 45 patients hair wash was one of the triggers and in 38 patients hair wash was a trigger concurrently and in combination with another common trigger. The effect of episodic and long-term prophylaxis in preventing this trigger-like headache has been analysed. The relieving factors were mostly rest and analgesic ingestion and sleep.

iv] Associated Clinical Conditions: In our study of 402 patients with migraine the following clinical conditions were seen associated – namely evidence of healed Granulomatous diseases, Seizures, Hypertension, Head Injury, Sinusitis, Psychiatric Changes and Stroke. The risk of stroke may be increased two- to threefold in migraine sufferers. Young adult sufferers and women using hormonal contraception appear to be at particular risk. The mechanism of this association though vastly unclear is attributed to chronic abnormalities of cerebral blood vessel tone [Etminan M et al 2005] [102]. Women who experience auras have been found to have twice the risk of strokes and heart attacks over non-aura migraine sufferers and women who do not have migraines [Kurth T et al 2006]

[103]. Similar observation was made in this study and all the patients who had migrainous strokes were female and all of them had migraine with aura. Migraine sufferers seem to be at risk for both thrombotic and hemorrhagic stroke as well as transient ischemic attacks [Becker C et al 2007] [104]

v] Special Types of Migraine: Our study also included 5 cases of Ophthalmoplegic Migraine of which 4 had Abducent Nerve palsy and 1 patient had Oculomotor Nerve palsy. This is a very rare condition in children, characterized by a migraine like attack Weiss AH et al [2006][105]. The oculomotor nerve is most commonly involved, with pupillary abnormality and ptosis, followed by the abducent, and rarely the trochlear nerve Ferrante E et al [2006][106]. The attack usually lasts from days to months and resolves spontaneously. A number of adult cases have been reported Lee TG et al [2002][107].

Twelve patients had basilar migraine and 10 out of 12 patients were in 1st and 2nd decade. The symptoms experienced by majority of them were vertigo, diplopia, ataxia, parasthesias, hemianopias, and decreased level of consciousness. Two of these patients also had some attacks without basilar features. These observations are in correlation with most international studies. Basilar migraine is common is adolescent age groups and more in males With increasing maturity of the nervous system attacks of basilar migraine become less common and generally are replaced by migraine without aura [Peatfield RC et al 2000] [108].

vi] Complication of Migraine: (a)Chronic Migraine: In our study of the 402 case of Migraine, 70(17.37%) patients had complications of migraine. Of these 70 patients, 46 patients had Chronic Migraine (9 patients of Chronic Migraine since Onset & 37 patients of Episodic Headache converting to Migraine), 5 patients had Migrainous Infarction (Cerebellar Infarction -3 & Right Occipital Infarction -2) and 19 patients had Migraine Trigerred Seizures (Migraine Terminating as Seizures – 14, Migralepsy - 5). No patients with Status Migrainous

or Persistent Aura without Infarction were included in the study. In our study, the most common complication observed in patients with migraine was transformation of migraine to chronic migraine or chronic daily headache. As the chronicity develops migraine headache lose its episodic presentation. Most of these patients with transformed migraine are patients with migraine without aura which is concordant with the studies of Siberstein SD et al 2001[109]. Five patients were diagnosed to have migrainous infarction in our study. All the five patients were females in the age group of 20-35 years. All the infarctions occurred in patients with migraine with aura and were in the posterior cerebral artery territory (3occipital infarcts & 2cerebellar infarcts). Women who experience auras have been found to have twice the risk of strokes and heart attacks over non-aura migraine sufferers and women who do not have migraines [Kurth T et al 2006][103].This observation in our study is also concordant with observation by Peter Goadsby P.J [2002][110]. Three patients had only migraine and no other risk factors. Two of these patients tested positive for anti-phospholipid antibodies. The incidence of stroke in this study is 0.9% as against 10-15% in the literature. This increased risk of stroke parallels the chronicity of migraine.

(b) Migraine Trigerred Seizures: In our study, we came across 61 instances of Migraine associated with Seizures, 19 patients had Migraine Triggered Seizures, 20 patients had Post Ictal Migrainous Headache and 22 patients had Migraine & Coexistent Seizure. Of the 19 patients who had Migraine Triggered Seizures, 14 patients had Migraine Terminating as Seizures and 5 patients had Migralepsy. Analysis of the 19 patients showed that 9 patients had GTCS, 5 patients had CPS and 5 patients presented with prolonged LOC. Migralepsy as defined in literature are seizures occurring during or within an hour of migrainous aura [Lennox WG et al 1960][111]. In our study of these 19 patients who had migraine which terminated as seizures, 14 patients differed from the classical description of migralepsy in their duration of headache with a window period of more than three hours well outside the

defined window period of 1 hour.. This group of 14 patients presented with both migraine with aura and migraine without aura. 12 of the 14 patients responded to anti-migrainous prophylaxis alone with a 1year episode free period, while 2 patients responded to anti-migrainous prophylaxis alone with a 3 & 6 month episode free period. Hence these patients were grouped as Migraine triggered seizures. These observations in our study correlate with observations of Anderman F et al (1987) [112].The timing and features of headache in patients with epilepsy were explored by various studies. Marks and Ehernberg et al [1993] [113] evaluated and established the relationship between catamenial epilepsy and patients with migraine with aura, showing an increased risk for an association between seizures and migraine. Lenaerts et al [1999][114] evaluated the degree of co-morbidity and established the pattern of temporal relationship between migraine and epilepsy in 202 patients in tertiary care clinics, which outlines that Migraine attacks equally precede or follow seizures, but migraine aura more often preceded the seizure. This correlated with the observations in our study. In analyzing our group of patients, migraine preceeded the seizures in 19 of 56 patients (14 migraine with aura), and followed it in 20 of 56 patients. Of the 19 patients, 5 patients had migralepsy and 14 patients had migraine which terminated as seizures. Relationship between duration of headache and terminations as seizures was analysed. In our patients, all had a prolonged duration of migraine ranging from 2->12hrs duration. This observation in our study is in contrast to the observation of Young et al [1983][115] in their epilepsy unit, where their patients had a brief duration of headache lasting for upto 20 minutes, which was of hemi-cranial throbbing pain, except for 2 patients who had a prolonged duration of headaches. Headache can also be the sole or most predominant manifestation of epileptic seizures, this is a relatively rare situation [Laplante P et al 1983][116]

(c) Migraine and Loss of Consciousness: Relation ship between Migraine & LOC was also analysed in our study group of migraine triggered seizures. In our study group 5 patients had

loss of consciousness. Episodes of impairment of consciousness have been reported in 10-20% of migraineurs in various series mainly in the young and the mechanisms is not clear. Sicuteri et al., found a marked hypersensitivity to bromocriptine in patients with presumptive migraine syncope. The drug caused marked hypotension in these patients, which was reversed by domperidone, suggesting dopamine receptor hypersensitivity. LOC in basilar migraine which is attributed to constriction of basilar artery can range from few minutes to 30 minutes. Stupurous periods in migraine (Migraine stupor and coma) vary from 12 hrs to 5 days during attacks of migraine. Measurement of cerebral blood flow during an episode of migraine stupor demonstrated a severe, global reduction in cerebral blood flow Bickerstaff ER et al 2005.[117].

B. TENSION TYPE HEADACHE In patients with TTH 18 presented with headache of more than 12hours duration, while 10 patients had a duration of 6-12 hours and 5 patients had a duration of 1-6hours. 31 of these patients experienced bilateral headaches with most (19 patients) confined to the frontal region. Bendtsen L et al [2006][118] evaluated and compiled the features of tension type headache. TTH is characterized by a bilateral, pressing, tightening pain of mild to moderate intensity, occurring in short episodes of variable duration (episodic forms) or continuously (chronic form). The headache is not associated with the typical migraine features, such as vomiting, severe photophobia, and phonophobia. In the chronic form, only one of these accompanying symptoms is allowed and only mild nausea is accepted. Because of lack of accompanying symptoms and milder pain intensity, patients rarely are severely incapacitated by their pain. TTH is the most featureless of the primary headaches and, because many secondary headaches may mimic TTH, a diagnosis of TTH requires exclusion of other organic disorders. A general and neurologic examination and prospective follow-up using diagnostic headache diaries6 with registration of all consumed drugs are, therefore, of utmost importance to reach a diagnosis. There are no reliable specific

paraclinical tests that are useful in differential diagnosis. Manual palpation of the pericranial muscles and their insertions should be done to demonstrate a possible muscular factor for patients and to plan treatment strategy, where physical training and relaxation therapy are important components. [Jensen R 1999][119].

The headache was holocranial in 31 of the 35 patients while it was hemicranial in the other 4 patients. Most of the patients experienced a aching type of headache (23 patients), while it was band like in 11 patients and throbbing in 6 patients. The predominant premonitary symptom was phonophobia (30 patients), with nausea, paraesthesia, giddiness and photophobia other common associated symptoms in order of occurrence. The most common aggravating factor in our study group was mental stress, while physical stress, travel, head bath and lack of sleep were also commonly reported. The relieving factors were mostly pressure and analgesics and coffee / tea ingestion. As TTH was commonly associated with depression and is aggravated by mental stress, previously was thought to be psychogenic now a neurological basis was established. Other common aggravating factors include poor self related health, inability to relax after work and sleeping few hours per night (Bendsten L etal 2000)[120]. In our study of 35 patients with TTH the following clinical conditions were seen associated – namely evidence of healed Granulomatous Diseases, Hypertension and Head Injury.

C. TENSION VASCULAR HEADACHE: Of the 78 patients diagnosed most patients had durations of more than 12 hours. 62 patients had an earlier diagnosis of Migraine without Aura whose frequency of episodes increased over a period of few years to chronic migraine / transformed migraine (more than 15 episodes per month) which progressed to bilateral holocranial headache with scalp tenderness in most patient. The episodic nature of migraine headache was lost and character of the headache changed, pattern shifting from typical throbbing pain to diffuse aching and band like pain with radiation to the neck. The

predominant accompanying symptoms like nausea, photophobia and phonophobia were less frequent and less intense in this group than compared to typical migraine. 15 patients of this group presented initially with episodic tension type headache later transforming to the mixed entity. The most common aggravating factor in our study group was mental stress and physical stress, travel, head bath and lack of sleep. The relieving factors were mostly analgesics, rest, and sleep. Of these 3 patients who were on multiple analgesics prescribed, medication overuse headache, responded after withdrawing them from drugs. In our study of 78 patients with TVH, 15 patients had associated mental depression. Majority of patients in this group required two drugs one antimigrainous prophylaxis and an added antidepressant for prevention or for reducing the intensity and severity of their headaches. This terminology has been used in our study as a separate entity, as these patients shared features of both migraine and Tension type headache. But Dodick et al 2006 [71] has considered the term Tension Vascular Headache antiquated.

D. TRIGEMINAL AUTONOMIC CEPHALAGIAS: In patients with TAC 9 presented with headache of less than 6 hours duration, while 2 patients had a duration of 6-12 hours. 2 of the patients experienced bilateral headaches while 9 had unilateral headaches with most (9 patients) confined to the frontal temporal region. Most of the patients experienced an aching type of headache (8 patients), while it was throbbing in 2 patients and pricking in 1 patient. The predominant premonitary symptom was nausea and vomiting (10 patients), with redness of eyes, nasal stuffiness, phonophobia, photophobia, drooping of eyelids and blurring of vision the other common associated symptoms in order of occurance. Aggravating factors were not reported in most patients in our study alcohol intake, sunlight and lack of sleep were reported. The releiving factors were mostly analgesics, pressure and coffee / tea ingestion. In consonance with our observations Rozen TD et al [1999][75] described the features of typical cluster headache. Typical cluster headache location is retro-orbital, periorbital, and

occipitonuchal. Maximum pain is normally retro-orbital in more than 70% of patients. Pain quality is described as boring, stabbing, burning, or squeezing. Cluster headache intensity is always severe and never mild, although headache pain intensity may be less at the beginning and end of cluster periods. The duration of individual cluster headaches is between 15 minutes and 180 minutes, with more than 75% attacks lasting less than 60 minutes. Attack frequency is between one and three attacks per day, with most patients experiencing two or fewer headaches in a day. Cluster headache is marked by its associated autonomic symptoms that typically occur on the same side as the head pain but can be bilateral. Lacrimation is the most common associated symptom, occurring in 73% of patients, followed by conjunctival injection in 60%, nasal congestion in 42%, nasal rhinorrhea in 22%, and a partial Horner's syndrome in 16% to 84%.

III DIAGNOSTIC STUDIES

A. Electrophysiology:

(i) Migraine: EEG Sharp waves and spikes in posterior occipital region mainly occipital region, more during the period of aura and was normal during the interval period between attacks of migraine. This correlates with a large multicenter study - incidence of spikes and paroxysmal events was 12.5% compared to 0.7% in normal adult volunteers. The percentage goes still high up in patients with seizures. Monomorphic or polymorphic slow waves, seen bilaterally. Few cases also showed slowing in posterior head region [Beaumanoir A et al 1987][121]. EEG was taken in 54 of the 402 patients with migraine 28 of the 54 patients showed Non Specific Slowing in Posterior Region, while 5 patients showed Spikes & Sharp Waves in Occipital Region while 21 of the patients had no specific changes

(ii) TTH EEG was taken in 12 of the 35 patients with TTH, 2 of these patients showed

Non Specific Slowing in Posterior Region, while 10 of the patients had no specific changes

(iii) TAC EEG was taken in 4 of the 11 patients with CH & TAC, all 4 patients showed no specific changes.

B. Autonomic Function Tests:

Based on a literature review and an extensive investigation of patients with migraine, Thomsen, Olesen and coworkers conclude that 'Clear dysfunction of the sympathetic nervous system remains to be shown. Mild parasympathetic hypofunction with denervation supersensitivity may be present in migraine' Results are variable. Boiardi et al.[1988][44] reported for instance that the diastolic blood pressure response to sustained handgrip was impaired in 61% of migraine patients. In our study (1) Low Frequency Heart Rate Variation (LF) with a mean control value of 40.34 and mean test value for migraineurs of 37.4 and a p value of 0.2837 was found not significant. The LF is mediated by sympathetic and parasympathetic contributions and is believed to reflect the vasomotor reflex (2) High Frequency Heart Rate Variation (HF) with a mean control value of 59.93 and a mean test value of 62.60 and p value of 0.620 was found not significant. The HF is mediated by parasympathetic nervous control and is related to respiratory sinus arrhythmia. Akselrod S et al [1981][122]. These parameters can be influenced by posture. (3) LF/ HF Ratio with a mean control value of 0.706 and a mean test value of 0.709 and p value of 0.975 was found not significant. Therefore, the LF/HF ratio reportedly represents sympathetic nervous control. However, Eckberg DL [1997][123] critically reviewed the simplified interpretation of the LF/HF ratio as an index of sympathovagal balance. Furlan et al [1999][124] observed significantly decreased LF power, increased HF power, and a decreased LF/HF ratio between midnight and 6 AM in ambulatory subjects (3) Mean Heart Rate with a mean control value of 74.0 and a mean test value of 76.20 and p value of 0.0327 was found significant. It is concluded that this value shows significant difference between the control and test. In a recent study by

Pierangeli G et al [1997][47] no heart-rate variability differences between migraine patients and control subjects were found (4) Mean RR Interval with a mean control value of 0.813 and a mean test value of 0.842 and p value of 0.738 was found not significant. Havanka-Kanniainen et al [1986][125] and Thomsen et al [1995][45] reported decreased R-R variations in subjects with migraine, which indicated parasympathetic hypofunction. Mikamo et al[1989] [126] and Pogacnik et al [1993][127] reported that there was no significant difference in R-R variability between migraineurs and controls. The findings of Masako T et al [2000][128] suggest that patients with migraine have cardiac parasympathetic hypofunction and abnormal rhythm generation.

B. Imaging Studies: (i) Migraine: CT scan of brain was taken in all of the 402 patients, of whom 83 had changes. The most common change reported in CT scan brain was calcified granulomas in 69 patients, gliosis in 8 patients and basal ganglia calcification in 6 patients. CT scan of the para-nasal sinuses was performed in 53 patients of which 18 showed maxillary sinusitis while it was normal in 35 patients. Frishberg BM et al 1994 [129] reviewed four CT scan studies, four MRI scan studies, and one combined MRI and CT scan study of 897 scans of patients who had migraine. These findings are combined with more recent reports of one CT scan study of 284 patients and six studies of MRI scans of 444 patients for a total of 1625 scans of patients who had various types of migraine. Other than white matter abnormalities, the studies showed no significant pathology except for four brain tumours (three of which were incidental findings) and one AVM (in a patient who had migraine and a seizure disorder). Sempere found a similarly low yield of 0.4%.

V. PROPHYLAXIS

A. MIGRAINE: The patients with migraine were given prophylactic therapy with either Propronolal (20-160mg) or Amitryptylline (12.5-50mg) or Propronolal (20-160mg) and Amitryptylline (12.5-50mg) or Flunarizine (5mg) or Topiramate (50mg) or Sodium Valproate ER(500mg). Patients with migraine with and without aura responded well to beta blocker at lower dosages with headache free interval of more than six months in majority. This is in correlation with meta analysis from many studies where beta blockers was associated with reduction in migraine activity and more than 100 clinical trials clearly established the benefits of propronolol. Patients with mixed tension vascular headache responded well to amitryptylline and a combination of beta blocker and amitryptylline than propronolol as evidenced in studies by Mathew NT et al [1991][130]. Flunarizine in doses of 5-10mg used in this study mainly adolescents and in patients with basilar features provided a good reduction in headache frequency and was well tolerated

B. TENSION TYPE HEADACHE: All patients in our study were treated with Amitryptiline (10-50mgms). Of the 35 patients 3 patients responded with a disease free interval of 1year, 9 patients with a disease free interval of 6 months to 1 year, 9 patients with a disease free interval of less than 6months, 11 patients responded with decreased intensity and frequency of episodes while 3 patients did not show any response. This is in concordance with various studies. Prophylactic pharmacotherapy include tricyclic antidepressant, amitriptyline, is the only drug proved effective in several controlled trials in TTH. Bendtsen L et al [2005][131]. The two most recent studies reported that amitriptyline (75 mg per day) reduced headache index (duration _ intensity) by 30% compared with placebo. The effect is long lasting (at least 6 months)55 and not related to the presence of depression. Bendtsen L et al [1996][132]. If patients do not respond to amitriptyline, mirtazapine could be attempted. Venlafaxine or SSRIs could be considered in patients who have concomitant depression, if tricyclics or mirtazapine are not tolerated.

C. TENSION VASCULAR HEADACHE: All 78 patients were treated with Amitryptiline (10-50mgms) and Propronolol (40-160mgms). Of the 78 patients 63 patients responded with decreased intensity and frequency of episodes while 15 patients did not show any response.

D. TRIGEMINAL AUTONOMIC CEPHALALGIAS: Two patients with SUNCT were on prophylaxis with sodium valproate with moderate response. Four patients with paroxysmal hemicrania responded well to Indomethacin during acute phases and were given prophylaxis with Amitrptiline with good response. All 4 patients had a disease free interval of six months. Prophylaxis was provided for 5 patients with cluster headache of which 3 were given Amitryptiline (25-50mgms), 2 patients were given Propranolol (80-120mgms). Of the 3 patients given Amitryptiline 2 patients responded with a disease free interval of 1year, 1 patient with a disease free interval of 6 months to 1 year. Of the 2 patients given Propranolol, both of them responded with a disease free interval of 1year. These drug therapies are at variance to usual protocols. But these patients showed marked responses indicating that these were probable Cluster Headache Migraine overlap syndromes. Solomon S et al [1986] [133] has referred to a similar clinical situation were the patients had clinical features of both types of headaches. But the criteria enunciated by him were considered imprecise. Graham et al [1975][134] referring to combined syndromes has described migraine headaches in recurrent bouts resembling cluster headache responding to migraine prophylaxis.

Summary

The observations of this study are here with summarised

1. Migraine is the commonest type of headache comprising of about 75%. Migraine without aura [48%] was more common than migraine with aura [32%]. Female preponderance was noticed in all subtypes of migraine, age of onset being in 2nd and 3rd decade for majority of the subgroups of migraine, except for basilar migraine which was common in 1st and 2nd decade.
2. Chronic migraine, Migraine triggered seizures and Migrainous infarction were the complications of migraine encountered in this study in the order of frequency of occurrence. Transformation to Chronic migraine was more common from episodic forms and in patients with onset of migraine in teens or twenties. Migrainous infarction was seen only in female patients who had exclusively migraine with aura and all the five patients had posterior circulation strokes.
3. 14 patients were grouped as migraine triggered seizures. These patients differed from the classical description of migralepsy in their duration of headache, with a window period of more than three hours prior to the seizure. This group of patients presented with both migraine with aura and migraine without aura and responded to anti-migrainous prophylaxis alone with a 1 year episode free period.
4. Mean Heart Rate shows difference between the control and migraine patients but is within physiological range and no variation was observed in power spectral analysis.
5. Patients with migraine with and without aura responded well to beta blocker at lower dosages with headache free interval of more than six months in majority.

Patients with basilar migraine benefited from either propronolal or flunarizine, but patients with chronic migraine responded predominantly to combination prophylaxis.

6. Patients with tension type headache responded to amitriptyline and those patients with tension vascular headache i.e. patients with features of tension type headache and of migraine responded well to a combination of beta blocker and amitryptyline.

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Appendix 1

PROFORMA FOR HEADACHE

Name: **Age:** **Sex:**

Residential address:

Occupation:

MIN No: **Headache clinic No**:

Details about headache: How long-
Age of onset-
Headache free period if any-

Duration in hours-

Location- Unilateral- Bilateral-

Radiation Temporal- to neck- Frontal - Retro orbital region- Occipital - Limbs (UL & LL) – Holocranial-

Character- Throbbing- Pricking- Tight sensation- Aching- Shock like- Jarring-

Frequency- Episodes/week- Episodes/month-
Recent change in frequency-
Frequency with prophylactic drugs-

Associated features- Photophobia and phonophobia-
Nausea and vomiting-
Yawning-
Drowsiness-
Giddiness/ vertigo-
LOC-
Redness of eyes-
Lacrimation-
Diplopia-
Parasthesias-

Aura- Visual- **Positive visual phenomena**: Small bright dots-
White spots/ flashes of light (photopsias)-
Fortification spectra (teichopsia)-
Other zig zag lines-
Coloured spots of light-

Negative visual phenomena: Blind spots (scotomas)-
Black dots/ spots-
Hemianopias-

Disturbances of visual perception- blurred vision-
Micropsia/ macropsia/teleopsia-
Tunnel vision-

Triggering factors- Physical stress/ mental stress- Sunlight-
Sleep alteration- Travel-
Headbath- Perfumes/fumes-
Cold items- Position of neck-
Others-

Relieving factors- Pressure- Coffee/ tea-
Analgesics Rest-.

Relationship to menstrual cycles-

Family history-

On Examination- BP- Temporal artery- Sinuses-

Palpabrel fissure- Refraction- Fundii-

Cranial nerves-

Long tract signs-

Associated conditions- Seizures- HT- Stroke-

Granulomatous diseases- Psychiatric diseases-

Investigation- CT scan Brain-

EEG (where required)

Diagnosis- **Primary**- **Secondary-**

Migraine / Tension type headache / Short lasting headache /

Cluster headache / Paroxysmal hemicrania

Medications- Propranolol Topiramate
Amitryptyline Flunarizine

Follow up-

Appendix 2

Master Charts

A. Migraine
B. Tension Type Headache
C. Cluster Headache
D. Tension Vascular Headache

APPENDIX 2 – A : MASTER CHART – MIGRAINE

AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	MONTHFREQUENCY /	AURA	FAM H	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBRE

AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	MONTHFREQUENCY /	AURA	FAM H	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE
25	F	7	Temporal	Throbbing	6	Visual	Yes			MWA	prop	DFI-12M
31	F	14	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+
17	M	15	Temporal	Pricking	4	Sensory&Visual	Yes			MWA	prop	DFI-6M+
23	F	30	Temporal	Throbbing	3			Calcif Granuloma		MWOA	Amit	DIF
8	F	3	Occipital	Aching	12		Yes			MWOA	Flunarazine	DFI-6M
50	F	24	Frontal	Throbbing	16					CM	amt + prop	DFI-6M+
25	F	8	Temporal	Throbbing	4	Visual	Yes			MWA-BM	prop	DFI-6M+
27	M	7	Temporal	Throbbing	6	Sensory	Yes			MWWOA	amt + prop	DFI-6M+
22	M	14	Temporal	Burning	5			Calcif Granuloma		MWOA	prop	DFI-6M+
18	F	4	Parietal	Throbbing	8					MWOA	prop	DFI-6M+
13	F	2	Occipital	Throbbing	4	Sensory & Visual	Yes			MWA-BM	Flunarazine	DFI-6M
15	M	8	Temporal	Pricking	2		Yes			MWOA	Amit	DIF
32	F	9	Parietal	Diffuse	6					MWOA	amt + prop	DFI-6M+
18	F	8	Frontal	Throbbing	2	Visual	Yes		GTCS	MTS	prop	DFI-6M+
44	F	14	Frontal	Throbbing	20					CM	amt + prop	DFI-6M+
23	M	20	Temporal	Throbbing	8	Visual		Calcif Granuloma		MWWOA	amt + prop	DFI-6M
25	F	4	Parietal	Throbbing	6	Visual	Yes			MWA	prop	DFI-6M+
14	F	8	Temporal	Aching	4		Yes			MWOA	Amit	DIF
25	F	15	Temporal	Aching	2	Sensory				MWWOA	amt + prop	DFI-6M+
10	F	5	Temporal	Throbbing	6		Yes	Calcif Granuloma		MWOA	Flunarazine	DFI-12M
42	F	8	Frontal	Throbbing	18					CM	amt + prop	DFI-6M+
33	F	14	Temporal	Throbbing	8	Visual	Yes			MWWOA	amt + prop	DFI-6M
22	M	5	Temporal	Pricking	2					MWOA	prop	DFI-6M+
29	M	8	Temporal	Pricking	4		Yes	Calcif Granuloma		MWOA	prop	DFI-6M
28	M	24	Temporal	Throbbing	6	Visual				MWWOA	amt + prop	DFI-6M+
15	F	4	Temporal	Aching	6		Yes			MWOA	prop	DFI-6M+
32	F	8	Frontal	Throbbing	2	Visual		Gliosis		MWWOA	amt + prop	DFI-6M
40	F	10	Frontal	Pricking	3		Yes			MWOA	prop	DFI-6M+
22	F	24	Temporal	Throbbing	4	Visual				MWA	prop	DFI-12M
25	F	5	Parietal	Throbbing	5		Yes			MWOA	Amit	DIF
20	F	12	Temporal	Throbbing	2	Visual	Yes		CPS	MTS	Sod Valp	DIF
18	M	7	Temporal	Throbbing	6	Visual	Yes		LOC	MTS	prop	DFI-12M
20	F	8	Temporal	Throbbing	8	Visual	Yes	Calcif Granuloma		MWA	amt + prop	DFI-6M+
27	M	24	Temporal	Pricking	10		Yes			MWOA	amt + prop	DFI-6M+
31	F	30	Frontal	Aching	12		Yes			MWOA	amt + prop	DFI-6M

ABBREVI

MWA – I
WITH AU
MWOA –
MIGRAI
WITHOU
MWWO
MIGRAI
OR WIT
AURA
CM – CH
MIGRAI
PM – PR
MIGRAI
MTS –M
TRIGER
SEIZUR
MIGSTR
MIGRAI
STROKE
BM – BA
MIGRAI
DFI-6M+
FREE IN
6MONTI
MONTH
DFI-6M
FREE IN
LESS TH
6MONTI
DFI-12M
FREE IN
MORET
MONTH
NR – NO
RESPO

AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	MONTHFREQUENCY /	AURA	FAM H	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE
22	F	7	Frontal	Throbbing	4	Visual	Yes			MWA	prop	DFI-12M
24	F	14	Temporal	Throbbing	2		Yes	Calcif Granuloma		MWOA	Amit	DIF
8	F	8	Occipital	Aching	2	Visual	Yes		LOC	MTS	Flunarazine	DFI-6M
36	F	15	Frontal	Throbbing	22					CM	amt + prop	DFI-6M+
14	F	9	Temporal	Pricking	4	sensory	Yes			MWWOA	amt + prop	DFI-6M+
27	M	24	Temporal	Diffuse	8			Calcif Granuloma		MWOA	Amit	DIF
35	F	14	Temporal	Throbbing	5					MWOA	prop	DFI-6M+
12	M	12	Temporal	Throbbing	4	Sensory & Visual	Yes			MWA-BM	prop	DFI-6M+
18	M	24	Frontal	Diffuse	11			Calcif Granuloma		MWOA	prop	DFI-6M+
36	F	15	Temporal	Throbbing	12					MWOA	Amit	DIF
13	M	7	Occipital	Throbbing	3	Visual	Yes			MWA-BM	Flunarazine	DFI-12M
29	F	12	Temporal	Throbbing	6	Visual		Gliosis	Stroke	MWA	amt + prop	DFI-6M+
14	M	14	Frontal	Throbbing	7					MWOA	amt + prop	DFI-6M
12	M	4	Temporal	Throbbing	2	sensory				MWWOA	amt + prop	DFI-6M+
38	F	7	Temporal	Throbbing	3		Yes		GTCS	MTS	prop	DFI-12M
16	F	3	Parietal	Pricking	5		Yes			MWOA	Amit	DFI-6M
40	F	12	Temporal	Throbbing	19		Yes			CM	amt + prop	DFI-6M
25	F	15	Temporal	Aching	7		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M+
18	M	13	Frontal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+
22	M	24	Frontal	Throbbing	4					MWOA	prop	DFI-6M+
38	F	12	Temporal	Aching	10		Yes			MWOA	Topiramate	DIF
12	M	14	Frontal	Throbbing	8			Basal Gang Calc		MWOA	Amit	DIF
40	F	24	Temporal	Throbbing	3	Visual	Yes			MWWOA	amt + prop	DFI-6M+
24	F	5	Temporal	Pricking	4					MWOA	amt + prop	DFI-6M
18	F	14	Temporal	Throbbing	2	Visual	Yes			MWA-BM	prop	DFI-6M+
27	M	15	Frontal	Throbbing	3		Yes		GTCS	MTS	Sod Valp	DFI-6M+
45	F	24	Temporal	Throbbing	19		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M+
16	F	6	Parietal	Aching	5		Yes			MWOA	Amit	DIF
15	F	12	Temporal	Throbbing	8	Visual	Yes			MWWOA	amt + prop	DFI-6M+
28	F	14	Temporal	Throbbing	11		Yes			MWOA	prop	DFI-6M+
14	M	7	Frontal	Aching	2	Visual			LOC	MTS	prop	DIF
15	F	15	Frontal	Throbbing	3	Visual	Yes			MTS	prop	DFI-12M
38	M	24	Temporal	Aching	20		Yes			CM	amt + prop	DFI-6M+
53	F	12	Temporal	Throbbing	18		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M
26	M	8	Frontal	Throbbing	3		Yes			MWOA	Amit	NR

ABBREVI

MWA – MIGRAINE AURA
MWOA – MIGRAINE WITHOUT
MWWOA MIGRAINE OR WITH AURA
CM – CHR MIGRAINE
PM – PRO MIGRAINE
MTS –MIG TRIGERR SEIZURES
MIGSTR– MIGRAIN STROKE
BM – BAS MIGRAINE
DFI-6M+ - DISEASE INTERVA 6MONTHS MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETHA MONTHS
NR – NO RESPONS

AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	MONTHFREQUENCY /	AURA	FAM H	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE
24	F	5	Frontal	Throbbing	8		Yes	Calcif Granuloma		MWOA	prop	DFI-6M+
32	F	24	Temporal	Pricking	2		Yes			MWOA	Amit	DIF
18	F	14	Temporal	Throbbing	4	Visual	Yes			MWA	prop	DFI-6M+
36	M	24	Temporal	Throbbing	15			Calcif Granuloma		CM	amt + prop	DFI-6M+
14	M	24	Temporal	Throbbing	3					MWOA	amt + prop	DFI-6M
17	F	4	Parietal	Burning	5		Yes			MWOA	amt + prop	DFI-6M+
14	M	5	Temporal	Throbbing	11	Sensory	Yes		CPS	MTS	prop	DFI-6M
18	F	16	Frontal	Throbbing	4		Yes			MWOA	prop	DFI-6M+
9	M	4	Occipital	Aching	2	Visual	Yes			MWA-BM	Flunarazine	DFI-6M
42	F	14	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+
26	F	12	Temporal	Throbbing	2	Visual	Yes			MWA	prop	DFI-6M
34	M	24	Temporal	Pricking	4		Yes	Calcif Granuloma		MWOA	Amit	DIF
24	F	12	Frontal	Throbbing	8					MWOA	prop	DFI-6M+
22	M	15	Temporal	Throbbing	6	Sensory & Visual	Yes	Gliosis		PM	amt + prop	DFI-6M
32	F	12	Temporal	Throbbing	5					MWOA	prop	DFI-6M+
14	F	8	Occipital	Aching	3	Visual	Yes			MWA-BM	Flunarazine	DFI-12M
45	F	24	Temporal	Throbbing	20			Calcif Granuloma		CM	amt + prop	DFI-6M+
28	M	12	Temporal	Pricking	8	Sensory	Yes			MWWOA	amt + prop	DFI-6M
24	M	12	Frontal	Throbbing	6		Yes			MWOA	amt + prop	DFI-6M+
26	F	24	Temporal	Throbbing	4	Visual	Yes			MWWOA	Topiramate	DIF
15	F	6	Temporal	Throbbing	2					MWOA	amt + prop	DFI-6M+
13	F	8	Occipital	Throbbing	5	Visual	Yes			MWA-BM	prop	DFI-6M
53	F	24	Temporal	Diffuse	18		Yes			CM	amt + prop	DFI-6M+
18	F	14	Temporal	Throbbing	3		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M+
38	M	12	Frontal	Throbbing	5		Yes			MWOA	Amit	DFI-6M
22	F	12	Temporal	Throbbing	2	Visual	Yes			MWA	prop	DFI-6M+
20	F	6	Occipital	Aching	1		Yes			MWOA	Flunarazine	DFI-12M
28	M	6	Temporal	Throbbing	8					MWOA	amt + prop	DFI-6M
36	F	3	Temporal	Throbbing	20	Visual	Yes			CM	amt + prop	DFI-6M+
34	M	12	Frontal	Throbbing	7	Visual				MWWOA	Amit	DFI-6M+
16	F	14	Frontal	Throbbing	4		Yes			MWOA	prop	DFI-6M+
18	F	8	Temporal	Pricking	8	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M
22	M	5	Temporal	Throbbing	5				GTCS	MTS	prop	DFI-6M+
20	F	8	Temporal	Pricking	3	Visual	Yes	Basal Gang Calc		MWWOA	amt + prop	DFI-6M+
19	F	10	Frontal	Throbbing	2	Visual	Yes			MWOA	Amit	DFI-6M

ABBREVI

MWA – MIGRAINE AURA
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DFI-6M+ - DISEASE INTERVA 6MONTHS MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETHA MONTHS
NR – NO RESPONS

18	F	5	Temporal	Throbbing	4	Visual	Yes			MWOA	amt + prop	DFI-6M+	**MWA** – MIGRAINE AURA
24	M	5	Temporal	Throbbing	2	Visual		Calcif Granuloma		MWA-BM	prop	DFI-6M	**MWOA** – MIGRAINE WITHOUT
16	F	12	Temporal	Throbbing	5		Yes			MWOA	amt + prop	DFI-6M	**MWWOA** MIGRAINE OR WITH AURA
42	F	12	Frontal	Throbbing	15		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M+	**CM** – CHR MIGRAINE
36	M	24	Temporal	Pricking	3		Yes			MWOA	amt + prop	DFI-6M+	**PM** – PRO MIGRAINE
15	F	5	Temporal	Throbbing	5		Yes			MWOA	prop	DFI-6M	**MTS** –MIG TRIGERR SEIZURES
24	F	7	Occipital	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M+	**MIGSTR**– MIGRAIN STROKE
28	F	12	Temporal	Throbbing	4		Yes	Calcif Granuloma		MWOA	Amit	DFI-6M+	**BM** – BAS MIGRAINE
32	M	6	Temporal	Throbbing	9					MWOA	prop	DFI-6M	**DFI-6M+** - DISEASE INTERVA 6MONTHS MONTHS
19	F	4	Frontal	Throbbing	12	Visual	Yes			MWWOA	amt + prop	DFI-6M+	**DFI-6M** - DISEASE INTERVA THAN 6M
14	F	2	Temporal	Pricking	4					MWOA	amt + prop	DFI-6M+	**DFI-12M** DISEASE INTERVA MORETH MONTHS
12	M	5	Frontal	Throbbing	4		Yes	Basal Gang Calc		MWOA	amt + prop	DFI-6M+	**NR** – NO RESPONS
25	F	6	Temporal	Throbbing	6	Sensory & Visual	Yes			MWWOA	prop	DFI-6M	
18	F	12	Temporal	Throbbing	5					MWA	Amit	DFI-12M	
20	M	3	Temporal	Pricking	4		Yes			MWOA	amt + prop	DFI-6M+	
22	F	14	Parietal	Throbbing	2	Visual	Yes			MWA	amt + prop	DFI-6M	
44	F	24	Temporal	Throbbing	20		Yes			CM	amt + prop	DFI-6M+	
18	F	9	Frontal	Throbbing	5			Calcif Granuloma		MWOA	prop	DFI-6M	
16	M	5	Temporal	Throbbing	3	Visual	Yes			MWWOA	amt + prop	DFI-6M+	
22	M	12	Temporal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+	
24	M	12	Temporal	Throbbing	8		Yes			MWOA	Amit	DFI-6M	
8	F	2	Temporal	Throbbing	12		Yes			MWOA	amt + prop	DIF	
29	F	4	Occipital	Aching	7	Visual	Yes			MWA	Flunarazine	DFI-6M	
19	F	24	Temporal	Throbbing	4		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M+	
17	F	12	Temporal	Throbbing	8					MWOA	amt + prop	DFI-6M+	
14	M	12	Frontal	Throbbing	5	Visual	Yes		CPS	MTS	prop	DFI-6M+	
25	F	12	Temporal	Pricking	3	Visual				MWWOA	amt + prop	DIF	
33	F	24	Parietal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M+	
39	M	24	Temporal	Throbbing	4		Yes			MWOA	prop	DFI-6M+	
43	F	6	Temporal	Throbbing	2					MWOA	amt + prop	DIF	
18	M	24	Temporal	Throbbing	20		Yes			MWOA	amt + prop	DFI-6M+	
20	F	12	Temporal	Throbbing	5	Visual	Yes	Gliosis	Stroke	CM	amt + prop	DIF	
26	F	4	Occipital	Aching	4	Visual	Yes			MWWOA	amt + prop	DFI-6M+	
23	F	3	Temporal	Burning	8		Yes			MWOA	prop	DFI-6M	
40	F	12	Frontal	Throbbing	5		Yes			MWOA	prop	DFI-6M	

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22	M	24	Temporal	Throbbing	3	Visual		Calcif Granuloma		MWA	amt + prop	DIF	
14	M	4	Temporal	Pricking	12	Visual	Yes			MWA	prop	DFI-6M	
18	F	2	Temporal	Throbbing	7					MWOA	amt + prop	DFI-6M+	
32	F	3	Temporal	Throbbing	14	Sensory	Yes			MWWOA	amt + prop	DFI-6M+	
20	F	8	Frontal	Throbbing	12		Yes			MWOA	prop	DFI-6M+	
45	F	5	Temporal	Throbbing	7	Visual	Yes	Calcif Granuloma		MWA	amt + prop	DIF	
26	M	15	Temporal	Throbbing	24		Yes			CM	amt + prop	DIF	
14	F	6	Temporal	Pricking	3		Yes			MWOA	Topiramate	DFI-6M	
18	F	2	Occipital	Aching	4	Visual	Yes			MWA-BM	Flunarazine	DFI-6M	
52	F	2	Temporal	Throbbing	12	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M+	
19	M	20	Temporal	Throbbing	12	Visual				MWWOA	amt + prop	DFI-6M+	
27	F	3	Occipital	Diffuse	16		Yes			CM	amt + prop	DFI-6M	
35	F	5	Temporal	Throbbing	12					MWOA	Amit	DFI-12M	
14	F	6	Frontal	Throbbing	12		Yes			MWOA	prop	DFI-6M+	
42	M	21	Temporal	Throbbing	24		Yes	Gliosis		CM	amt + prop	DFI-6M+	
18	F	3	Temporal	Throbbing	8	Visual				MWWOA	Sod Valp	DFI-6M+	
20	F	3	Temporal	Pricking	12		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-12M	
38	M	5	Temporal	Throbbing	12		Yes			MWOA	amt + prop	DIF	
25	M	8	Parietal	Throbbing	24		Yes			MWOA	Amit	DIF	
19	F	11	Temporal	Pricking	8		Yes			MWOA	amt + prop	DFI-6M+	
12	F	5	Occipital	Throbbing	5	Sensory & Visual	Yes			MWA-BM	prop	DFI-6M+	
18	F	4	Temporal	Throbbing	12			Calcif Granuloma		MWOA	amt + prop	DFI-12M	
42	M	18	Frontal	Pricking	24		Yes			CM	amt + prop	DFI-6M+	
20	F	3	Temporal	Burning	8	Visual			GTCS	MTS	prop	DFI-6M	
36	M	2	Temporal	Throbbing	14	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	
24	F	3	Temporal	Throbbing	12	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M+	
26	F	4	Frontal	Throbbing	12					MWOA	prop	DFI-6M	
30	F	5	Temporal	Pricking	24		Yes			MWOA	Amit	DFI-6M+	
34	F	3	Frontal	Throbbing	12		Yes			MWOA	amt + prop	DFI-6M+	
22	F	4	Frontal	Throbbing	12	Visual	Yes			MWA	prop	DFI-12M	
18	M	8	Temporal	Throbbing	6		Yes			MWOA	amt + prop	DIF	
15	F	11	Occipital	Pricking	4		Yes			MWOA	Flunarazine	DFI-12M	
18	F	2	Temporal	Throbbing	14	Visual				MWWOA	amt + prop	DFI-6M+	
36	M	8	Temporal	Pricking	24		Yes			MWOA	Sod Valp	DFI-6M+	
29	M	6	Frontal	Throbbing	12			Basal Gang Calc		MWOA	prop	DFI-6M	

MWA – MIGRAINE AURA
MWOA – MIGRAINE WITHOUT
MWWOA MIGRAINE OR WITH AURA
CM – CHR MIGRAINE
PM – PRO MIGRAINE
MTS –MIG TRIGERR SEIZURES
MIGSTR– MIGRAIN STROKE
BM – BAS MIGRAINE
DFI-6M+ - DISEASE INTERVA 6MONTHS MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETH MONTHS
NR – NO RESPONS

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14	F	5	Temporal	Burning	24	Visual	Yes			MWA	prop	DFI-6M+
18	M	12	Occipital	Diffuse	3		Yes			MWOA	amt + prop	DFI-6M+
22	F	14	Temporal	Throbbing	3	Visual	Yes	Calcif Granuloma		MWA	amt + prop	DIF
34	F	12	Frontal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+
19	M	24	Temporal	Aching	8		Yes			MWWOA	prop	DFI-6M+
43	F	24	Temporal	Pricking	12					CM	amt + prop	DFI-12M
52	F	7	Occipital	Throbbing	7		Yes			MWOA	Amit	DFI-6M+
38	F	12	Temporal	Throbbing	4					MWOA	amt + prop	DFI-12M
24	M	8	Frontal	Throbbing	8		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M+
22	F	14	Temporal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+
19	F	12	Frontal	Aching	3					MWOA	Amit	DFI-12M
17	F	24	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M+
29	F	12	Parietal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M
51	M	7	Temporal	Throbbing	2	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF
36	F	12	Parietal	Throbbing	20		Yes			CM	amt + prop	DFI-12M
18	M	8	Temporal	Diffuse	5		Yes			MWOA	prop	DFI-6M+
26	M	24	Frontal	Throbbing	4	Visual				MWWOA	amt + prop	DIF
22	F	5	Occipital	Throbbing	8		Yes			MWOA	Amit	DIF
21	F	12	Temporal	Throbbing	5					MWOA	Topiramate	DFI-6M
19	M	7	Temporal	Aching	3		Yes			MWOA	amt + prop	DIF
24	F	12	Frontal	Throbbing	12			Calcif Granuloma		MWOA	amt + prop	DIF
35	M	24	Temporal	Pricking	7	Visual	Yes			MWWOA	amt + prop	DFI-6M+
16	F	24	Temporal	Throbbing	14			Calcif Granuloma		MWOA	prop	DFI-6M+
17	F	6	Occipital	Pricking	1	Visual	Yes			MWWOA	Flunarazine	DFI-12M
14	M	12	Temporal	Throbbing	3					MWOA	amt + prop	DFI-6M+
50	M	12	Temporal	Throbbing	16		Yes			CM	amt + prop	DIF
39	M	8	Frontal	Throbbing	20					CM	amt + prop	DIF
14	F	24	Frontal	Throbbing	3	Sensory				MWA	prop	DFI-6M+
11	F	14	Temporal	Throbbing	22		Yes			MWOA	Amit	DFI-12M
26	F	7	Temporal	Throbbing	20	Visual	Yes	Gliosis	Stroke	CM	amt + prop	DFI-6M
32	F	24	Occipital	Burning	2	Sensory	Yes			MWWOA	Flunarazine	DIF
28	M	24	Frontal	Throbbing	8		Yes			MWOA	amt + prop	DFI-6M+
17	F	24	Temporal	Burning	6	Visual	Yes		GTCS	MTS	prop	DFI-6M+
15	F	8	Temporal	Throbbing	2					MWOA	amt + prop	DFI-6M+
32	F	9	Parietal	Throbbing	4	Visual	Yes			MWA	prop	DFI-6M

MWA – MIGRAINE AURA
MWOA – MIGRAINE WITHOUT
MWWOA MIGRAINE OR WITH AURA
CM – CHR MIGRAINE
PM – PRO MIGRAINE
MTS –MIG TRIGERR SEIZURES
MIGSTR– MIGRAIN STROKE
BM – BAS MIGRAINE
DFI-6M+ - DISEASE INTERVA 6MONTHS MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETHA MONTHS
NR – NO RESPONS

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21	M	12	Temporal	Diffuse	4	Visual	Yes			MWWOA	amt + prop	DFI-6M+	**MWA** – MIGRAINE AURA
17	F	12	Frontal	Throbbing	8		Yes	Calcif Granuloma		MWOA	prop	DFI-6M	**MWOA** – MIGRAINE WITHOUT
8	F	24	Temporal	Aching	5	Visual		Calcif Granuloma		MWA	prop	DFI-6M	**MWWOA** MIGRAINE OR WITH AURA
13	F	6	Occipital	Throbbing	3		Yes			MWOA	prop	DFI-6M	**CM** – CHR MIGRAINE
8	F	12	Occipital	Diffuse	2	Sensory	Yes			MWWOA	Flunarazine	DFI-6M+	**PM** – PRO MIGRAINE
15	F	6	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	**MTS** –MIG TRIGERR SEIZURES
29	F	24	Temporal	Throbbing	2		Yes			MWOA	prop	DIF	**MIGSTR**– MIGRAIN STROKE
31	F	24	Frontal	Throbbing	24	Visual	Yes			CM	amt + prop	DFI-6M+	**BM** – BAS MIGRAINE
29	M	12	Frontal	Throbbing	3	Visual	Yes			MWWOA	amt + prop	DFI-12M	**DFI-6M+** - DISEASE INTERVA 6MONTHS MONTHS
15	F	24	Temporal	Pricking	2			Calcif Granuloma		MWOA	amt + prop	DFI-6M+	**DFI-6M** - DISEASE INTERVA THAN 6M
18	M	6	Temporal	Burning	4		Yes			MWOA	amt + prop	DFI-6M	**DFI-12M** DISEASE INTERVA MORETH MONTHS
44	F	6	Parietal	Throbbing	2					MWOA	prop	DFI-6M+	**NR** – NO RESPONS
20	F	6	Parietal	Throbbing	5		Yes			MWOA	amt + prop	DFI-6M	
24	M	24	Temporal	Throbbing	15		Yes			CM	amt + prop	DFI-6M	
15	F	12	Occipital	Pricking	4					MWOA	Amit	NR	
51	M	12	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
26	F	12	Temporal	Aching	5	Visual	Yes		LOC	MTS	prop	DFI-12M	
25	F	24	Occipital	Throbbing	3		Yes			MWOA	prop	DFI-6M+	
30	F	12	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M	
32	F	24	Frontal	Throbbing	18		Yes			CM	amt + prop	DFI-12M	
18	F	12	Temporal	Pricking	8					MWOA	amt + prop	DFI-6M	
47	M	12	Temporal	Throbbing	6		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	
22	M	14	Frontal	Throbbing	2	Visual				MWWOA	amt + prop	DFI-12M	
34	F	6	Temporal	Throbbing	4	Visual	Yes		CPS	MTS	prop	DFI-6M+	
37	F	24	Temporal	Aching	15		Yes			CM	amt + prop	DFI-6M	
12	F	6	Temporal	Throbbing	8		Yes			MWOA	Amit	DFI-6M	
21	F	12	Frontal	Throbbing	6		Yes			MWOA	amt + prop	DFI-12M	
25	F	6	Frontal	Throbbing	8	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M	
29	F	6	Occipital	Aching	6		Yes			MWOA	Flunarazine	DFI-12M	
15	F	24	Temporal	Throbbing	2	Visual	Yes	Gliosis	Stroke	MIGSTR	prop	DFI-6M+	
19	F	12	Frontal	Throbbing	4					MWOA	amt + prop	DFI-6M	
38	F	24	Temporal	Throbbing	3		Yes			MWOA	Amit	DFI-6M+	
22	M	6	Temporal	Throbbing	2	Sensory				MWWOA	Topiramate	DFI-6M	
18	M	6	Temporal	Pricking	4		Yes	Basal Gang Calc		MWOA	prop	DFI-6M+	
35	F	12	Parietal	Throbbing	2	Visual	Yes			MWOA	Amit	NR	

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34	M	24	Temporal	Diffuse	2		Yes			MWOA	prop	DFI-6M	**MWA** – MIGRAINE AURA
24	F	12	Temporal	Pricking	5	Visual				MWA	amt + prop	DFI-6M	**MWOA** – MIGRAINE WITHOUT
35	F	24	Frontal	Throbbing	20	Visual	Yes			CM	amt + prop	DFI-12M	**MWWOA** MIGRAINE OR WITH AURA
9	F	12	Frontal	Throbbing	6		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	**CM** – CHR MIGRAINE
16	M	6	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-12M	**PM** – PRO MIGRAINE
28	F	6	Temporal	Pricking	6					MWOA	amt + prop	DFI-6M	**MTS** –MIG TRIGERR SEIZURES
18	F	12	Occipital	Aching	6	Visual	Yes			MWWOA	amt + prop	DFI-6M	**MIGSTR**– MIGRAIN STROKE
22	M	12	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M	**BM** – BAS MIGRAINE
20	F	6	Frontal	Throbbing	6		Yes			MWOA	amt + prop	DIF	**DFI-6M+** - DISEASE INTERVA 6MONTHS MONTHS
32	F	12	Parietal	Throbbing	2		Yes		GTCS	MTS	Sod Valp	DFI-6M	**DFI-6M** - DISEASE INTERVA THAN 6M
25	M	6	Temporal	Aching	4	Visual	Yes			MWWOA	Amit	DFI-12M	**DFI-12M** DISEASE INTERVA MORETH MONTHS
17	M	24	Temporal	Throbbing	3		Yes			MWOA	amt + prop	DFI-6M	**NR** – NO RESPONS
35	M	6	Parietal	Throbbing	15			Calcif Granuloma		CM	amt + prop	DFI-6M	
17	F	6	Temporal	Throbbing	2	Sensory	Yes	Calcif Granuloma		MWWOA	prop	DIF	
19	F	6	Temporal	Throbbing	6					MWOA	Flunarazine	DIF	
25	F	24	Occipital	Aching	6		Yes			MWOA	amt + prop	DFI-12M	
42	F	12	Frontal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
29	F	12	Temporal	Throbbing	6	Visual				MWWOA	prop	DFI-12M	
12	F	24	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
44	F	126	Frontal	Throbbing	18		Yes	Calcif Granuloma		CM	prop	DIF	
17	M	24	Temporal	Burning	2	Sensory & Visual	Yes			MWWOA	Flunarazine	DFI-6M+	
22	M	6	Occipital	Throbbing	3		Yes			MWOA	amt + prop	DFI-12M	
28	F	6	Frontal	Throbbing	15		Yes			MWOA	amt + prop	DFI-6M	
36	F	12	Temporal	Pricking	5	Visual				MWWOA	Amit	NR	
19	F	24	Occipital	Throbbing	3		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-12M	
24	F	6	Temporal	Throbbing	2					MWOA	amt + prop	DFI-6M	
20	M	12	Frontal	Throbbing	5		Yes			MWOA	Flunarazine	DIF	
28	F	12	Temporal	Aching	5	Visual	Yes			CM	amt + prop	DFI-6M	
22	F	24	Temporal	Pricking	3		Yes			MWOA	amt + prop	DFI-12M	
18	M	12	Occipital	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
18	F	24	Temporal	Throbbing	20	Visual	Yes			MWWOA	amt + prop	DIF	
17	M	6	Temporal	Aching	5		Yes		LOC	MTS	prop	DFI-6M	
21	F	24	Frontal	Throbbing	3		Yes			MWOA	amt + prop	DFI-12M	
17	M	12	Parietal	Throbbing	5	Visual		Calcif Granuloma		MWA	prop	DIF	
32	F	12	Temporal	Pricking	3		Yes			MWOA	Amit	DFI-6M+	

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34	F	6	Temporal	Pricking	2					MWOA	Amit	DFI-6M	
7	F	12	Temporal	Pricking	6	Visual				MWA	prop	DIF	
18	F	12	Frontal	Throbbing	15		Yes	Calcif Granuloma		CM	amt + prop	DFI-12M	
24	F	24	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M	
44	F	6	Occipital	Aching	6	Visual	Yes			MWWOA	Flunarazine	DFI-12M	
15	M	6	Temporal	Throbbing	3		Yes			MTS	prop	DIF	
36	F	12	Frontal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
29	F	24	Frontal	Throbbing	18		Yes			CM	amt + prop	DFI-6M	
27	M	6	Occipital	Diffuse	6	Visual	Yes			MWWOA	prop	DIF	
32	M	12	Temporal	Throbbing	5					MWOA	Amit	DFI-12M	
18	F	6	Parietal	Throbbing	6		Yes	Basal Gang Calc		MWOA	amt + prop	DFI-6M	
50	F	6	Temporal	Throbbing	6	VS				MWA	prop	DIF	
15	M	24	Temporal	Aching	3		Yes			MWOA	amt + prop	DFI-12M	
17	F	6	Frontal	Throbbing	6		Yes			MWOA	amt + prop	DFI-6M	
38	M	24	Frontal	Throbbing	20		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M	
22	M	6	Occipital	Throbbing	4	Sensory				MWWOA	amt + prop	DFI-12M	
24	F	12	Temporal	Pricking	6		Yes			MWOA	amt + prop	DFI-6M	
36	F	24	Temporal	Throbbing	2	Visual	Yes			MWWOA	Amit	NR	
52	F	12	Temporal	Throbbing	11	Sensory	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M	
32	F	12	Temporal	Throbbing	6	Visual		Gliosis	Stroke	MIGSTR	prop	DIF	
28	F	24	Frontal	Aching	8		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	
18	M	12	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	
35	F	12	Temporal	Pricking	18		Yes			CM	amt + prop	DFI-6M	
28	M	6	Temporal	Throbbing	1	Visual	Yes			MWWOA	amt + prop	DFI-6M	
19	F	6	Temporal	Burning	4		Yes			MWOA	prop	DFI-12M	
15	F	24	Frontal	Throbbing	3		Yes	Calcif Granuloma		MWOA	Amit	DFI-6M	
53	F	12	Occipital	Throbbing	6	Visual				MWWOA	Amit	DFI-6M	
27	M	24	Temporal	Throbbing	3		Yes			MWOA	prop	DIF	
15	F	24	Frontal	Throbbing	4					MWOA	amt + prop	DFI-6M	
33	F	24	Temporal	Throbbing	22	Visual	Yes			CM	amt + prop	DFI-12M	
16	M	12	Temporal	Throbbing	6		Yes			MWOA	Amit	DFI-6M+	
19	F	6	Occipital	Aching	4	Visual		Calcif Granuloma	GTCS	MTS	prop	DIF	
26	F	24	Parietal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-12M	
18	F	6	Temporal	Pricking	2		Yes			MWOA	amt + prop	DFI-6M	
18	M	24	Temporal	Aching	6	Visual	Yes	Calcif Granuloma		MWA	prop	DIF	

MWA – MIGRAINE AURA
MWOA – MIGRAINE WITHOUT
MWWOA MIGRAINE OR WITH AURA
CM – CHR MIGRAINE
PM – PRO MIGRAINE
MTS –MIG TRIGERR SEIZURES
MIGSTR– MIGRAIN STROKE
BM – BAS MIGRAINE
DFI-6M+ - DISEASE INTERVA 6MONTHS MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETHA MONTHS
NR – NO RESPONS

AGE
SEX
DURATION IN HRS
LOCATION
CHARACTER
MONTHFREQUENCY /
AURA
FAM H
CTSCAN - BRAIN
COMPLICATIONS
DIAGNOSIS
PROPHYLAX
RESPONSE
ABBREVI

10	F	12	Temporal	Throbbing	4		Yes			MWOA	Amit	DFI-6M+	**MWA** – MIGRAINE AURA
36	F	6	Temporal	Aching	3	Visual	Yes	Calcif Granuloma		MWA	prop	DIF	**MWOA** – MIGRAINE WITHOUT
35	M	24	Frontal	Aching	15		Yes			CM	amt + prop	DFI-12M	**MWWOA** MIGRAINE OR WITH AURA
18	F	6	Occipital	Throbbing	2	Visual	Yes			MWWOA	Flunarazine	DFI-6M+	**CM** – CHR MIGRAINE
42	F	24	Frontal	Throbbing	5					MWOA	amt + prop	DIF	**PM** – PRO MIGRAINE
16	F	12	Temporal	Pricking	3		Yes	Calcif Granuloma		MWOA	Amit	DFI-12M	**MTS** –MIG TRIGERR SEIZURES
30	M	6	Temporal	Throbbing	2	Sensory & Visual		Calcif Granuloma		MWA	amt + prop	DFI-6M	**MIGSTR**– MIGRAIN STROKE
20	M	24	Frontal	Aching	8		Yes			MWOA	amt + prop	DFI-12M	**BM** – BAS MIGRAINE
52	F	6	Temporal	Throbbing	6	Sensory	Yes			MWWOA	amt + prop	DFI-6M	**DFI-6M+** - DISEASE INTERVA 6MONTHS MONTHS
18	F	12	Parietal	Throbbing	4		Yes			MWOA	prop	DIF	**DFI-6M** - DISEASE INTERVA THAN 6M
16	F	24	Temporal	Pricking	8	Visual				MWWOA	Amit	DFI-6M	**DFI-12M** DISEASE INTERVA MORETH MONTHS
24	M	24	Frontal	Throbbing	18		Yes	Calcif Granuloma		CM	amt + prop	DFI-12M	**NR** – NO RESPONS
40	F	6	Parietal	Throbbing	10		Yes			MWOA	amt + prop	DFI-6M	
25	M	6	Temporal	Throbbing	4	Visual	Yes			MWWOA	amt + prop	DFI-6M	
30	F	12	Temporal	Aching	2	Visual				MWA	prop	DIF	
26	M	12	Frontal	Throbbing	4		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-12M	
51	F	5	Frontal	Throbbing	20		Yes			CM	amt + prop	DFI-6M	
20	F	12	Temporal	Throbbing	2		Yes			MWOA	prop	DIF	
22	F	24	Temporal	Aching	3	Visual	Yes		CPS	MTS	prop	DIF	
18	F	24	Occipital	Diffuse	2		Yes			MWOA	Amit	DFI-6M+	
12	M	6	Temporal	Throbbing	1		Yes			MWOA	prop	DFI-12M	
19	M	12	Temporal	Throbbing	4					MWOA	amt + prop	DFI-6M	
26	F	12	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DIF	
33	F	24	Frontal	Throbbing	15					CM	amt + prop	DIF	
15	F	6	Occipital	Pricking	2		Yes			MWOA	Flunarazine	DFI-6M+	
27	M	6	Occipital	Throbbing	3	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	
35	F	12	Temporal	Burning	4	Sensory				MWWOA	amt + prop	DIF	
40	M	24	Occipital	Throbbing	4		Yes			MWOA	amt + prop	DFI-6M	
18	F	24	Temporal	Throbbing	6		Yes			MWOA	prop	DIF	
25	F	12	Temporal	Pricking	8	Visual	Yes			MWOA	Amit	DFI-6M+	
23	M	12	Frontal	Aching	16		Yes			CM	amt + prop	DFI-6M	
35	F	24	Temporal	Throbbing	8		Yes			MWOA	amt + prop	DFI-12M	
26	F	12	Frontal	Throbbing	8		Yes			MWOA	Amit	DFI-6M	
22	F	6	Frontal	Aching	4		Yes	Calcif Granuloma		MWA	prop	DIF	
25	F	24	Parietal	Throbbing	3	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	

AGE
SEX
DURATION IN HRS
LOCATION
CHARACTER
MONTHFREQUENCY /
AURA
FAM H
CTSCAN - BRAIN
COMPLICATIONS
DIAGNOSIS
PROPHYLAX
RESPONSE
ABBREVI

17	F	6	Temporal	Pricking	2		Yes	Calcif Granuloma		MWOA	amt + prop	DIF	
28	F	24	Frontal	Throbbing	5	Visual	Yes			MWA	prop	DFI-12M	
14	M	6	Parietal	Throbbing	3		Yes			MWOA	Flunarazine	DFI-6M+	
32	F	24	Temporal	Throbbing	2	Sensory & Visual	Yes			MWA	prop	DIF	
20	F	12	Temporal	Pricking	8		Yes			MWOA	amt + prop	DFI-12M	
26	M	24	Frontal	Throbbing	6		Yes			MWOA	Amit	DFI-6M+	
17	M	6	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DIF	
19	F	6	Temporal	Burning	8					MWOA	prop	DIF	
24	F	24	Temporal	Throbbing	7	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	
28	F	12	Temporal	Throbbing	9			Calcif Granuloma		MWOA	Amit	DFI-6M+	
15	F	6	Occipital	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	
26	F	24	Temporal	Pricking	2	Visual	Yes			MWWOA	amt + prop	DIF	
19	F	6	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DIF	
30	F	24	Temporal	Aching	11					MWOA	amt + prop	DFI-12M	
25	M	12	Frontal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DIF	
12	F	6	Occipital	Throbbing	3	Sensory	Yes			MWA-BM	prop	DIF	
38	M	24	Temporal	Diffuse	20		Yes			CM	amt + prop	DIF	
15	F	6	Temporal	Throbbing	1	Visual				MWWOA	prop	DFI-12M	
24	F	12	Frontal	Throbbing	4		Yes			MWOA	amt + prop	DIF	
28	F	24	Temporal	Aching	2	Visual	Yes			MWWOA	amt + prop	DFI-12M	
26	F	24	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	
40	M	24	Temporal	Pricking	8		Yes	Calcif Granuloma		MWOA	Amit	DFI-12M	
40	F	6	Parietal	Throbbing	7		Yes			PM	Flunarazine	DFI-6M+	
18	F	6	Temporal	Throbbing	9	Visual	Yes			MWWOA	amt + prop	DIF	
14	F	24	Temporal	Throbbing	4	Visual	Yes			MWWOA	amt + prop	DFI-12M	
26	F	12	Frontal	Aching	4					MWOA	amt + prop	DIF	
19	M	12	Temporal	Throbbing	8		Yes			MWOA	amt + prop	DFI-12M	
22	F	6	Temporal	Burning	7					MWOA	amt + prop	DIF	
20	F	6	Temporal	Pricking	9		Yes			MWOA	amt + prop	DIF	
28	F	6	Occipital	Throbbing	4	Sensory	Yes			MWWOA	Flunarazine	DFI-12M	
13	M	24	Frontal	Throbbing	8		Yes			MWOA	amt + prop	DIF	
30	F	6	Occipital	Throbbing	7	Sensory		Calcif Granuloma		MWWOA	Flunarazine	DFI-6M+	
15	F	6	Temporal	Pricking	9		Yes			PM	amt + prop	DIF	
19	M	24	Frontal	Pricking	4	Visual	Yes			MWOA	Amit	DFI-6M+	
27	F	12	Frontal	Throbbing	2		Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	

MWA – MIGRAIN AURA
MWOA – MIGRAIN WITHOU
MWWOA MIGRAIN OR WITH AURA
CM – CH MIGRAIN
PM – PR MIGRAIN
MTS –MI TRIGERR SEIZURE
MIGSTR– MIGRAIN STROKE
MWA-BM BASILLA MIGRAIN
DFI-6M+ DISEASE INTERVA 6MONTH MONTHS
DFI-6M - DISEASE INTERVA THAN 6M
DFI-12M DISEASE INTERVA MORETH MONTHS
NR – NO RESPON

DURATION IN HRS	LOCATION	CHARACTER	MONTHFREQUENCY /	AURA	FAM H	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBRI
12	Parietal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DIF	**MWA** – MIGRA
6	Frontal	Throbbing	14		Yes			MWOA	amt + prop	DFI-12M	**MWOA** – MIGR
24	Temporal	Pricking	3	Visual	Yes	Calcif Granuloma		MWWOA	prop	DIF	AURA
6	Temporal	Throbbing	9	Sensory & Visual	Yes			MWWOA	amt + prop	DFI-6M	**MWWOA** – MI
24	Temporal	Aching	10		Yes			MWOA	amt + prop	DIF	WITHOUT AUR
12	Frontal	Throbbing	12					PM	amt + prop	DFI-6M	**CM** – CHRONI
6	Occipital	Throbbing	4		Yes			MWOA	prop	DIF	**PM** – PROBAB
24	Occipital	Pricking	5	Sensory				MWWOA	Flunarazine	DFI-6M+	**MTS** –MIGRAI
6	Frontal	Throbbing	12		Yes			MWOA	amt + prop	DFI-12M	SEIZURES
24	Frontal	Throbbing	8		Yes			MWOA	amt + prop	DIF	**MIGSTR**– MIG
12	Temporal	Burning	12					MWOA	amt + prop	DIF	**BM** – BASILLA
6	Temporal	Throbbing	7	Sensory	Yes			MWWOA	amt + prop	DFI-12M	**DFI-6M+** - DIS
24	Occipital	Burning	2	Visual	Yes			MWWOA	Flunarazine	DFI-6M+	INTERVAL 6M
6	Temporal	Aching	4		Yes			MWOA	amt + prop	DIF	MONTHS
12	Frontal	Throbbing	4		Yes			MWOA	prop	DFI-12M	**DFI-6M** - DISE
24	Parietal	Throbbing	7		Yes	Calcif Granuloma		PM	Amit	DIF	INTERVAL LES
24	Temporal	Pricking	24	Sensory	Yes			CM	amt + prop	DFI-12M	**DFI-12M** DISE INTERVAL MO MONTHS **NR** – NO RESP

APPENDIX 2 – B : MASTER CHART – TENSION TYPE HEADACHE

AGE	SEX	DURATION IN HRS	LOCATION	HOLO /HEMI CRANIAL	CHARACTER	MONTHFREQUENCY /	FAM H	SCALP TEND	CTSCAN - BRAIN	DIAGNOSIS	PROPHYLAX	Dose in mg	RESPONSE	ABBRE

35	F	3	TEMPORAL	HEMI	THROBBING	4	YES	YES	calcific granuloma	IETTH	Amitryptiline	10	DFI-6M+
29	F	8	OCCIPITAL	HOLO	BAND	3		YES		IETTH	Amitryptiline	10	DIF
48	M	16	FRONTAL	HOLO	PRICKING	10	YES		calcific granuloma	FETTH	Amitryptiline	25	DFI-6M
34	M	4	TEMPORAL	HOLO	ACHE	16	YES	YES		CTTH	Amitryptiline	50	DFI-12M
36	F	14	OCCIPITAL	HOLO	ACHE	3		YES		PTTH	Amitryptiline	25	DFI-6M+
43	F	7	FRONTAL	HOLO	BAND	11		YES	calcific granuloma	FETTH	Amitryptiline	25	DIF
22	F	15	FRONTAL	HOLO	ACHE	12	YES		calcific granuloma	IETTH	Amitryptiline	10	DIF
18	F	9	PARIETAL	HEMI	THROBBING	17		YES	calcific granuloma	CTTH	Amitryptiline	50	DFI-6M
38	F	18	TEMPORAL	HOLO	ACHE	20	YES	YES		CTTH	Amitryptiline	25	DFI-6M+
45	F	36	TEMPORAL	HOLO	BAND	9		YES		IETTH	Amitryptiline	10	NR
27	M	20	FRONTAL	HOLO	ACHE	20		YES	Gliosis	CTTH	Amitryptiline	50	DFI-6M
53	F	5	OCCIPITAL	HOLO	ACHE	15	YES			CTTH	Amitryptiline	25	DIF
37	F	10	FRONTAL	HOLO	BAND	10		YES		FETTH	Amitryptiline	25	NR
28	F	24	FRONTAL	HOLO	BAND	5		YES	calcific granuloma	PTTH	Amitryptiline	10	DIF
33	M	24	FRONTAL	HOLO	THROBBING	15				CTTH	Amitryptiline	25	DFI-6M+
56	F	4	FRONTAL	HOLO	THROBBING	8	YES			FETTH	Amitryptiline	25	DFI-6M+
44	F	24	PARIETAL	HOLO	PRICKING	16	YES		Gliosis	CTTH	Amitryptiline	50	DFI-12M
26	F	7	TEMPORAL	HEMI	ACHE	18		YES		CTTH	Amitryptiline	25	DIF
42	F	22	FRONTAL	HOLO	BAND	15		YES		CTTH	Amitryptiline	25	DIF
38	M	9	FRONTAL	HOLO	BAND	2	YES	YES	calcific granuloma	PTTH	Amitryptiline	25	DFI-6M+
29	M	3	FRONTAL	HOLO	BAND	20				CTTH	Amitryptiline	50	DFI-6M
43	F	32	FRONTAL	HOLO	ACHE	17		YES		CTTH	Amitryptiline	25	DFI-6M+
38	F	20	OCCIPITAL	HOLO	PRICKING	3		YES	calcific granuloma	IETTH	Amitryptiline	10	DFI-6M
25	F	7	TEMPORAL	HOLO	ACHE	27	YES			CTTH	Amitryptiline	50	DFI-12M
44	F	18	TEMPORAL	HOLO	BAND	22		YES		CTTH	Amitryptiline	25	DIF
47	F	24	FRONTAL	HOLO	ACHE	10			calcific granuloma	FETTH	Amitryptiline	25	DFI-6M
42	F	13	FRONTAL	HOLO	THROBBING	24		YES		CTTH	Amitryptiline	25	DFI-6M+
36	F	6	FRONTAL	HOLO	ACHE	4	YES	YES		PTTH	Amitryptiline	10	DIF
45	M	24	PARIETAL	HOLO	PRICKING	2		YES	Gliosis	IETTH	Amitryptiline	10	DFI-6M
45	F	8	FRONTAL	HEMI	ACHE	16		YES		CTTH	Amitryptiline	25	DFI-6M+
17	F	24	FRONTAL	HOLO	BAND	7			calcific granuloma	FETTH	Amitryptiline	25	DIF
39	F	18	FRONTAL	HOLO	BAND	15		YES	calcific granuloma	CTTH	Amitryptiline	50	DFI-6M
26	M	10	TEMPORAL	HOLO	ACHE	17	YES	YES		CTTH	Amitryptiline	25	DIF
44	F	24	FRONTAL	HOLO	PRICKING	19				CTTH	Amitryptiline	25	DFI-6M
39	M	11	OCCIPITAL	HOLO	THROBBING	21		YES	calcific granuloma	CTTH	Amitryptiline	25	NR

IETTH – INFRE
EPISO

FETTH – FREQU
EPISO

CTTH – CHRO

PTTH – PROBA

DIF – DECRE
INTEN
FREQU

DFI-6M – DISEA
INTER
6MONT
MONT

DFI-6M – DISEA
INTER
THAN

DFI-12 – DISEA
INTER
MORE
MONT

NR – N
RESPO

APPENDIX 2 – C : MASTER CHART – TRIGEMINAL AUTONOMIC CEPHALAGIAS

SEX	AGE OF ONSET	DURATION IN HRS	LOCATION	CHARACTER	ASSOCIATION	TRIGGER	FAM H	CTSCAN - BRAIN	DIAGNOSIS	PROPHYLAX	Dose in mg	RESPONSE
	39	11	FT	ACHE	AUTONOMIC FR +		-	NORMAL	CCH	Amitryptiline	25	DFI-12M
	32	6	FT	ACHE	AUTONOMIC FR +	ALCOHOL	-	NORMAL	ECH	Amitryptiline	50	DFI-12M
	36	5MIN	FT	PRICK	AUTONOMIC FR +	SUNLIGHT	-	NORMAL	SUNCT	Carb + prop	50	DFI-12M
	22	6	P	THROBBING	AUTONOMIC FR +		-	NORMAL	EPHC	Propranolol	80	DFI-12M
	35	8	FT	ACHE	AUTONOMIC FR +	ALCOHOL	-	NORMAL	ECH	Amitryptiline	50	DFI-6M+
	49	6	FT	ACHE	AUTONOMIC FR +	ALCOHOL	-	NORMAL	CCH	Propranolol	120	DFI-12M
	37	3	FT	ACHE	AUTONOMIC FR +		-	NORMAL	CPHC	Amitryptiline	50	DFI-12M
	30	6	FT	ACHE	AUTONOMIC FR +	LACK OF SLEEP	-	NORMAL	ECH	Amitryptiline	25	DFI-12M
	27	5MIN	FT	ACHE	AUTONOMIC FR +		-	NORMAL	SUNCT	Carb +Prop	80	DFI-12M
	35	12	P	ACHE	AUTONOMIC FR +		-	NORMAL	CPHC	Amitryptiline	50	DFI-12M
	38	3	FT	THROBBING	AUTONOMIC FR +	LACK OF SLEEP	-	NORMAL	EPHC	Amitryptiline	50	DFI-12M

ABBR

CCH – CH
CLUSTER

ECH –EP
CLUSTER

EPHC – E
PAROXY
HEMICRA

CPHC – C
PAROXY
HEMICRA

DFI-6M+ –
FREE INT
6MONTH

DFI-12M –
INTERVA
12 MONT

APPENDIX 2 – D : MASTER CHART – TENSION VASCULAR HEADACHE

AGE	SEX	DURATION IN HRS	CHARACTER	MONTHFREQUENCY /	FAM H	CTSCAN - BRAIN	PFRESENTING DIAGNOSIS	DIAGNOSIS	SPECIFIC	PROPHYLAX		RESPONSE	ABBREVIAT
										AMITRY PTILINE	PROPRONOLOL		
33	F	7	Band Like	6	Yes		TTH	TVH		25	120	DFI-12M	**TVH - TENS VASCULAR HEADACHE**
42	F	14	Throbbing	20	Yes		Migraine	TVH		25	100	DFI-6M+	**DFI-6M+** - DISEASE FR INTERVAL 6MONTHS - MONTHS
44	F	13	Pricking	4		Calcif Granuloma	Migraine	TVH		25	80	DFI-6M	**DFI-6M** - DISEASE FR INTERVAL L THAN 6MON
36	M	12	Band Like	3	Yes		Migraine	TVH		25	100	DFI-12M	**DFI-12M** DISEASE FR INTERVAL MORETHAN MONTHS
32	F	24	Band Like	12	Yes		TTH	TVH		50	80	DFI-6M+	**NR** – NO RESPONSE
40	F	18	Aching	16			Migraine	TVH		25	120	DFI-6M+	
48	M	14	Diffuse	4			Migraine	TVH		25	80	DFI-6M+	
52	M	14	Diffuse	6			Migraine	TVH		25	80	DFI-6M+	
33	M	4	Throbbing	5	Yes		Migraine	TVH		25	80	DFI-6M+	
55	F	12	Aching	8	Yes		Migraine	TVH		25	80	DFI-6M+	
47	F	12	Band Like	4	Yes		Migraine	TVH		25	100	DFI-12M	
33	M	19	Diffuse	2			TTH	TVH		25	80	DFI-12M	
37	M	18	Diffuse	6	Yes		Migraine	TVH		25	80	DFI-6M	
32	F	36	Throbbing	2	Yes		Migraine	TVH		25	100	DFI-6M+	
43	F	20	Aching	20			Migraine	TVH		50	100	DFI-6M+	
38	M	24	Aching	8	Yes		Migraine	TVH		25	100	DFI-6M+	
47	F	18	Band Like	6	Yes		Migraine	TVH		25	80	DFI-6M	
54	M	15	Band Like	4			TTH	TVH	Medication Overuse	25	100	DFI-12M	
50	F	15	Diffuse	2	Yes	Calcif Granuloma	Migraine	TVH		25	80	DFI-12M	
31	F	28	Throbbing	6	Yes		Migraine	TVH		25	80	DFI-6M	
44	M	14	Band Like	18			Migraine	TVH		50	100	DFI-12M	
39	M	5	Aching	8			Migraine	TVH		25	80	DFI-12M	
36	F	24	Throbbing	2	Yes		Migraine	TVH		25	80	DFI-6M	
32	F	24	Band Like	4			TTH	TVH		25	80	DFI-6M	
37	F	24	Diffuse	6	Yes		Migraine	TVH		25	80	DFI-6M+	
40	M	12	Diffuse	6		Gliosis	Migraine	TVH		25	120	DFI-6M	
45	F	36	Band Like	2	Yes		TTH	TVH		25	80	DFI-6M+	
51	F	24	Aching	3	Yes		Migraine	TVH		50	80	DFI-6M	
37	F	15	Aching	4			Migraine	TVH		25	80	DFI-12M	
35	F	12	Band Like	5	Yes		TTH	TVH	Medication Overuse	25	120	DFI-12M	
40	M	12	Diffuse	2	Yes		TTH	TVH		25	80	DFI-6M	
35	F	20	Throbbing	6	Yes	Calcif Granuloma	Migraine	TVH		25	100	DFI-6M	
42	F	24	Diffuse	8			Migraine	TVH		25	120	DFI-6M+	
33	M	30	Diffuse	10	Yes		Migraine	TVH		50	100	DFI-6M	
37	F	7	Band Like	12	Yes		TTH	TVH		50	100	DFI-6M+	

AGE	SEX	DURATION IN HRS	CHARACTER	MONTHFREQUENCY /	FAM H	CTSCAN - BRAIN	PFRESENTING DIAGNOSIS	DIAGNOSIS	SPECIFIC	PROPHYLAX		RESPONSE	ABBREVIAT
										AMITRY PTILINE	PROPRONOLOL		

36	M	36	Throbbing	4	Yes		Migraine	TVH		25	80	DFI-6M	**TVH - TENS VASCULAR HEADACHE**
54	F	24	Diffuse	8			TTH	TVH		50	80	DFI-6M+	**DFI-6M+** - DISEASE FR INTERVAL 6MONTHS - MONTHS
36	F	9	Band Like	2	Yes		Migraine	TVH		25	80	DFI-6M+	**DFI-6M** - DISEASE FR INTERVAL L THAN 6MON
33	M	22	Band Like	22	Yes		Migraine	TVH		25	120	DFI-6M	**DFI-12M** DISEASE FR INTERVAL MORETHAN MONTHS
33	M	24	Aching	4			Migraine	TVH		25	80	DFI-12M	**NR** – NO RESPONSE
42	F	24	Throbbing	8	Yes		Migraine	TVH		25	80	DFI-12M	
44	F	14	Aching	5			Migraine	TVH		25	100	DFI-12M	
44	M	24	Diffuse	4	Yes		TTH	TVH		25	80	DFI-6M	
36	F	24	Diffuse	11			Migraine	TVH		25	100	DFI-6M	
42	F	12	Band Like	12	Yes		Migraine	TVH		25	100	DFI-12M	
40	F	12	Aching	3		Gliosis	Migraine	TVH		25	80	DFI-6M	
54	F	14	Throbbing	6			Migraine	TVH		25	80	DFI-6M	
50	F	4	Diffuse	7	Yes		Migraine	TVH		25	80	DFI-6M+	
31	F	6	Diffuse	2			Migraine	TVH		25	80	DFI-6M	
44	F	36	Band Like	3	Yes		TTH	TVH		50	100	DFI-6M+	
49	M	14	Diffuse	5			Migraine	TVH		50	80	DFI-6M	
36	F	15	Band Like	19	Yes		Migraine	TVH		25	120	DFI-12M	
32	F	12	Band Like	7	Yes		TTH	TVH		25	80	DFI-12M	
37	F	24	Throbbing	5			Migraine	TVH		25	80	DFI-6M	
40	F	12	Band Like	4	Yes		Migraine	TVH		25	80	DFI-12M	
45	F	14	Aching	10	Yes		Migraine	TVH		25	100	DFI-12M	
52	M	24	Band Like	8	Yes		TTH	TVH		25	80	DFI-6M+	
43	F	30	Diffuse	3			Migraine	TVH		25	80	DFI-6M+	
47	F	24	Diffuse	4	Yes		Migraine	TVH		25	100	DFI-6M+	
35	M	5	Aching	2	Yes		Migraine	TVH		25	80	DFI-12M	
40	F	36	Throbbing	3	Yes	Calcif Granuloma	Migraine	TVH		25	120	DFI-12M	
35	F	24	Band Like	19	Yes		TTH	TVH	Medication Overuse	25	80	DFI-12M	
42	F	24	Band Like	5			Migraine	TVH		25	80	DFI-6M+	
33	M	30	Aching	8	Yes		Migraine	TVH		25	80	DFI-6M+	
37	F	24	Diffuse	11	Yes		Migraine	TVH		25	80	DFI-12M	
36	F	30	Diffuse	2	Yes		Migraine	TVH		25	120	DFI-12M	
42	F	24	Aching	3			Migraine	TVH		25	80	DFI-6M	
46	F	12	Band Like	20	Yes		Migraine	TVH		25	80	DFI-12M	
33	F	18	Band Like	18	Yes		TTH	TVH		25	80	DFI-12M	
33	F	15	Band Like	3	Yes		Migraine	TVH		25	80	DFI-6M	

NO	AGE	SEX	DURATION IN HRS	CHARACTER	MONTHFREQUENCY /	FAM H	CTSCAN - BRAIN	PFRESENTING DIAGNOSIS	DIAGNOSIS	SPECIFIC	PROPHYLAX		RESPONSE	ABBREVIATIONS
											AMITRY PTILINE	PROPRO NOLOL		
71	42	F	24	Diffuse	8			Migraine	TVH		50	80	DFI-6M+	**TVH - TENSION VASCULAR HEADACHE**
72	34	F	24	Throbbing	2			Migraine	TVH		25	80	DFI-6M+	**DFI-6M+** - DISEASE FREE INTERVAL 6MONTHS -12 MONTHS
73	44	F	24	Aching	4	Yes		Migraine	TVH		25	80	DFI-6M+	**DFI-6M** - DISEASE FREE INTERVAL LESS THAN 6MONTHS
74	36	F	24	Diffuse	15	Yes		Migraine	TVH		25	120	DFI-6M	**DFI-12M** DISEASE FREE INTERVAL MORETHAN 12 MONTHS
75	32	F	14	Diffuse	3	Yes		Migraine	TVH		25	80	DFI-6M+	**NR** – NO RESPONSE
76	33	F	15	Diffuse	5	Yes		Migraine	TVH		25	80	DFI-6M+	
77	36	F	12	Diffuse	11	Yes		Migraine	TVH		25	80	DFI-6M+	
78	41	F	24	Aching	4			Migraine	TVH		25	100	DFI-6M	